University of Bath

DOCTOR OF HEALTH (DHEALTH)

A comparison of usual care physiotherapy, a pedometer-based walking intervention and a combination of both to treat patients suffering from nociceptive or neuropathic chronic lower back pain: A Randomised Controlled Trial

Feher, Richard

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A comparison of usual care physiotherapy, a pedometer-based walking intervention and a combination of both to treat patients suffering from nociceptive or neuropathic chronic lower back pain:

A Randomised Controlled Trial

Richard Feher

A thesis submitted for the degree in Doctorate in Health

University of Bath

Department of Health

July 2020

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Signature of author.....Richard Feher

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Abstract

Chronic lower back pain (CLBP) remains a physiotherapy treatment challenge with evidence lacking in which treatments best improve outcomes. Furthermore, the presence of CLBP phenotypes, including nociceptive and neuropathic phenotypes, is rarely mentioned in physiotherapy literature.

The primary objective of this randomised controlled trial was to assess changes in pain intensity between baseline and 12-week follow-up, between and within the following three treatment groups: usual care physiotherapy (P), a partly supervised pedometer-based walking intervention (W), and a combination of both (PW) in patients with nociceptive or neuropathic CLBP. Secondary objectives assessed changes in disability, kinesiophobia and pain catastrophizing between baseline and 12-week follow-up between and within the three groups.

The review of literature demonstrates the complex neurophysiology involved in CLBP pain phenotypes. Physiotherapists currently lack a comprehensive knowledge of pain. Associated psychosocial pain outcomes in literature exploring usual care physiotherapy and walking has been sparse. The limited randomized controlled trials involved up until now have not fully explored walking as exercise independently nor combined with usual care physiotherapy to treat CLBP.

A sample of 147 participants, 62.6% (92/147) female and 37.4% (55/147) males; mean age (SD) 46.2 (10.9) years with nociceptive (52.4%, 77/147) or neuropathic (47.6%, 70/147) CLBP were recruited from three private practice physiotherapy clinics in Johannesburg, South Africa. Consenting participants completed self-reported measures of pain intensity, disability, kinesiophobia, pain catastrophizing. Physical activity was measured using pedometers to record weekly steps. Participants were randomly allocated to P (n=46), W (n=52), or PW (n=49) groups, and followed up at 12-weeks (completion 72.8%, 107/147).

An intention-to-treat analysis using a linear mixed model showed significant improvement in pain intensity ($p<0.01$), disability ($p<0.01$), kinesiophobia ($p<0.01$) and pain catastrophizing ($p<0.01$) in all groups but there was no statistically significant difference between groups at 12-week follow-up. However, a minimally clinically important difference in pain intensity was only observed in the PW group at the 12-week follow-up. Moreover, greater than two physiotherapy visits showed a significant improvement in pain intensity ($p=0.01$), kinesiophobia ($p=0.01$) and on pain catastrophizing ($p=0.01$). Further exploration of the ideal number of physiotherapy visits may be necessary to improve outcomes optimally.

In conclusion, no statistically significant difference was found between the three treatments investigated.

List of Abbreviations

ACSM	American College of Sports Medicine
ANOVA	Analysis of variance
BMI	Body mass index
CBT	Cognitive behavioural therapy
CLBP	Chronic lower back pain
CNS	Central nervous system
CONSORT	Consolidated Standards of Reporting Trials
DNIC	Diffuse noxious inhibitory controls
EIM	Exercise is Medicine
FABQ	Fear avoidance behaviour questionnaire
fMRI	Functional magnetic resonance imaging
GABA	Gamma-Aminobutyric Acid
IASP	International Association for the Study of Pain
IHME	Institute for Health Metrics and Evaluation
IMMPACT	Initiative on Methods, Measurement, and Pain Assessment in clinical trials
LBP	Lower back pain
LMM	Linear mixed model
MCID	Minimally clinically important difference
MRI	Magnetic resonance imaging
NHS	National Health Service
NICE	National Institute for Health and Care Excellence
NP	Nociceptive pain
NSCLBP	Non-specific chronic lower back pain
NSAIDS	Non-steroidal anti-inflammatories
NRS	Numerical rating scale of pain
NeuPSIG	Special Interest Group on Neuropathic Pain
ODI	Oswestry Disability Index
P	Usual care (physiotherapy)
PW	Usual care (physiotherapy) and pedometer-based walking intervention
PCS	Pain Catastrophizing Scale
PCOQ	Patient Centred Outcome Questionnaire
PGAP	Progressive Goal Attainment Program
PNE	Pain Neuroscience Education
PNP	Neuropathic (peripheral) pain
PA	Physical activity

RCT	Randomised Controlled Trial
RMDQ	Roland Morris Disability Questionnaire
SA	South Africa
TKS	Tampa Scale for Kinesiophobia
TENS	Transcutaneous electrical nerve stimulation
UK	United Kingdom
VAS	Visual Analogue Scale
WHO	World Health Organization
W	Pedometer-based walking intervention

Chapter 1: Introduction

1.1 Research overview

Internationally, Lower Back Pain (LBP) is estimated to affect up to 85% of individuals during their lifetime (van Tulder et al., 2006a; Rubin, 2007). In developed countries, it is estimated to affect 84% of working adults (Thiese et al., 2014). Up to 15% of individuals experiencing LBP progress to experiencing chronic lower back pain, (CLBP) (Cilliers and Maart 2013). CLBP diagnosis is based on anatomical location and a timeframe of LBP lasting for more than three-months, unlike acute LBP which normally resolves in six-weeks (Liddle, Baxter and Gracey, 2004).

Using World Health Organization data, it has been predicted that from 2010-2020, developing countries are likely to experience the greatest prevalence of CLBP (at least one episode in a lifetime) (Woolf and Pfleger, 2003). A recent systematic review concurs with this in emphasizing that contributing factors are likely to be biopsychosocial in nature (Meucci, Fassa and Faria, 2015). These include low income and lower levels of education both associated with poorer living and working conditions. (Woolf and Pfleger, 2003; Meucci, Fassa and Faria, 2015). Two systematic reviews reporting on prevalence in CLBP and LBP in Africa concur that these factors may consequently cause these populations to experience the greatest impact on their economic, societal, and public health when compared with developed nations (Louw, Morris and Grimmer-Somers, 2007; Morris et al., 2018).

The diagnosis of LBP is mainly characterised by the presence of pain (Goldby et al., 2006). Difficulties with comparing pain prognoses in patients with LBP include different aetiologies, treatments, and outcome parameters (Rubin, 2007). Methods used to determine a favourable prognosis are variable, it may be defined by complete amelioration of pain, return to work or discontinued treatment. As a source of treatment, sign and symptom, pain itself has not received enough attention in physiotherapy studies. Unlike the 2009 UK NICE guidelines, the 2016 UK NICE guidelines do encourage patient stratification using the STarT back tool highlighting the involvement of pain phenotypes and biopsychosocial risk factors (National Collaborating Centre for Primary Care [UK], 2009; National Institute for Health and Care Excellence, 2016). Pain is often considered a symptom of pathology, but it has been described as a disease, with emotional, sensory, and cognitive consequences (Meyer, 2007). Often the origin of pain in CLBP is unknown and some researchers argue that effective management requires more research into defining pain phenotypes within cohorts (Chetty et al., 2012; Baron et al., 2016; Courtney, Fernández-de-Las-Peñas and Bond, 2017). Pain phenotypes classify pain according to the dominant neurophysiological mechanism responsible for

its origin and/ or maintenance (Smart, O'Connell and Doody 2008; Courtney, Fernández-de-Las-Peñas and Bond, 2017). Where pain once was considered as a homogenous entity, CLBP has been described as heterogonous, occurring in distinct phenotypes, largely accepted as having a major neuropathic or nociceptive component (Smart et al., 2012a; Smart et al., 2012b; Baron et al., 2016; Spahr et al., 2017). Nociceptive pain (NP) can arise from noxious stimulation of the lumbar spine or associated soft tissues (Costigan, Scholz and Woolf, 2009; Hoheisal et al., 2013). Neuropathic (peripheral) pain (PNP) has been explained as a lesion/ dysfunction of the somatosensory system (Woolf, 2004). Up until now, the literature reveals pain phenotypes have not been explored in a modelling process when examining patients with CLBP in physiotherapy studies.

Physiotherapy treatment is common practice in conservative therapy for CLBP (Qaseem et al., 2017). Over the last three decades, physiotherapy treatments have evolved from a biomedical to a biopsychosocial approach, with the former concentrating on mechanical treatments and the latter integrating biological, psychological, and social components (Gatchel et al., 2007). Conservative therapy or non-surgical CLBP management includes traditional medical management e.g., cupping, pharmacotherapy, self-management programs e.g., strength and stretching exercises and interventional pain management e.g., rhizotomies (Airaksinen, et al 2006; Chou et al, 2007; Krein et al., 2013). The UK National Institute for Health and Care Excellence's (NICE) recommendations for CLBP management, parallel those used by physiotherapists in South Africa (National Collaborating Centre for Primary Care [UK], 2009; Naidoo et al., 2012). Both recommend the combined use of massage, manipulation, exercise, and advice on self-management e.g., remaining physically active.

Exercise is a sub-set of physical activity and is advocated in the treatment of CLBP (Chou, 2010). There is still debate regarding the optimal type, frequency, and duration of exercise (Lawford, Walters and Ferrar, 2015). As yet, with isometric lumbar stabilizing exercises and walking programs advised with varied methodologies, an ideal exercise program that is superior to others is yet to be found. Physical activity (PA) and exercise in the form of walking have been recently explored as a conservative treatment for CLBP (Lawford, Walters and Ferrar., 2015; Sitthipornvorakul et al., 2018). However, its usefulness as an optimal treatment for CLBP is equivocal when compared to standard treatments utilised by physiotherapists such as varied amounts of spinal manipulation, massage, electrotherapy, and isometric lumbar stabilizing exercises (Hendrick et al., 2010; Lawford, Walters and Ferrar., 2015; Sitthipornvorakul et al., 2018).

Treatments using usual care physiotherapy, walking exercise and a combination of the two have not yet been compared. The current trial proposed to use a randomised controlled study design to investigate the effect of usual care physiotherapy modalities, a walking intervention, and a

combination of both on the biopsychosocial outcomes of CLBP, including pain intensity, disability, kinesiophobia, and catastrophic thinking. It will examine these in the context of nociceptive and neuropathic pain phenotypes in the modelling process.

1.2 Research question and aim

Research question: Does a usual care physiotherapy intervention or a combination of usual care physiotherapy and a partly supervised pedometer-based walking intervention show greater improvements in pain intensity and associated CLBP outcomes than a partly supervised pedometer-based walking intervention alone in participants with chronic low back pain? Walking programs used in previous studies have not been compared to usual care physiotherapy intervention or a combination of usual care physiotherapy and a partly supervised pedometer-based walking intervention whilst objectively measuring steps taken in all treatment arms. The treatments in this trial have never all been compared to each other in a single trial.

Aim: To determine whether a 12-week pedometer-based walking intervention versus usual care (comprising of manipulation, massage, and isometric lumbar stabilization exercises) or a combination of the two interventions, improves participants' perception of pain intensity, disability, kinesiophobia and pain catastrophizing, in relation to CLBP.

1.3 Statement of purpose

Conservative treatments for CLBP are poorly understood including usual care physiotherapy and walking exercise which have been used previously as treatments. The two have not yet been combined and trialled against its constituent parts in a study. This trial sets out to investigate the difference between treatments including a partly supervised walking program, usual care physiotherapy, and a combination of both on participants with CLBP using appropriate biopsychosocial outcome measures. In addition, pain has been considered as a homogenous entity previously, pain phenotyping categorizing nociceptive and neuropathic pain phenotypes was included in the current modelling process. In order to accomplish the trial, it was necessary to achieve the following two objectives:

Objectives:

a. To determine whether a pedometer-based walking intervention, usual care, or a combination of the two best decreases pain intensity at 12-week follow-up.
b. To determine whether a pedometer-based walking intervention, usual care, or a combination of the two best decreases disability, kinesiophobia and pain catastrophizing at 12-week follow-up.

1.4 Organisation of thesis

A search strategy was conducted prior to the literature review. Listed sources included were PubMed, Web of science, Scopus, Embase and Google. The search was structured using studies with keywords of chronic lower back pain, treatment, massage, manipulation, physiotherapy, walking, pedometer, exercise, physical activity, neurophysiology, pain phenotype, nociceptive pain, neuropathic pain, lumbar spine. Each search used a combination of free text and subject headings. The search combined these keywords so that studies were identified that included chronic lower back pain and treatment, and in addition had terms relating to either walking or physiotherapy treatment. Only randomized controlled trials using walking and physiotherapy relevant to lower back pain lasting for longer than three months were included in the review. Treatment was furthermore searched relating to nociceptive or neuropathic pain phenotypes. The listed journals were searched from first issue 1960 to March-April 2020.

Following an introduction in chapter one, an in-depth review of the relevant literature was undertaken in chapter two. This was followed by a methodology including a feasibility study described in chapter three. The methodology was set out to justify the explicit design of three treatment groups due to the heterogenous nature of treatments used in previous studies using walking and physiotherapy to treat CLBP. Screening tools are discussed to familiarize readers with pain phenotyping using the painDETECT questionnaire. All the outcome measures are described in this section. To improve objective step measurement between treatment groups, pain and activity diaries are well described which were used by all participants and not only those using the pedometer-based walking intervention. A feasibility study was conducted prior to the main RCT to streamline trial processes used if necessary. The methodology describes the main RCT which compared a 12-week pedometer-based walking intervention versus usual care (comprising of manipulation, massage and isometric lumbar stabilization exercises) versus a combination of the two interventions. The reference group used in this RCT was the pedometer-based walking intervention. The term usual care physiotherapy was used since usual care is a term used to describe “the full spectrum of patient care practices in which clinicians have the opportunity (which is not necessarily seized) to individualize care” (Thompson and Schoenfeld, 2007). The participants (n=147) were Johannesburg citizens with CLBP

randomised to the three treatment groups having equal numbers of nociceptive and neuropathic CLBP participants in each treatment group. Comparisons were conducted on participants' perception of pain intensity, disability, kinesiophobia and pain catastrophizing, in relation to nociceptive and neuropathic CLBP. Results in chapter four from the main RCT include linear mixed models and descriptive analysis of the data. Chapter five is a discussion of the interpretation of the results from the main RCT. The relevance of the findings is discussed in the context of the aim and objectives posed at the start of this thesis and in relation to the previous literature. Limitations of the current RCT are discussed with recommendations made for future study for clinical use. Chapter six is the conclusion of the thesis with insights into potential relevance of the study findings to the clinical environment. The references are followed by supplementary information provided in the appendices.

1.5 Conflict of interest statement

Fifty OMRON Walking Style One 2.1 HJ 321 E Pedometers were purchased and 30 were supplied as a sponsorship from Omron Europe. The sponsored pedometers were obtained six-weeks after the RCT began therefore there was no conflict of interest. The author declares that he has neither competing interests nor conflict of interest.

Chapter 2: Review of the Literature:

2.1 Epidemiology of chronic lower back pain

Internationally, LBP is one of the most common and frequent medical conditions among adults (Schwellnus et al., 2011). LBP is defined as pain between the inferior ribcage and above the inferior gluteal fold. It may present with or without leg pain (Duffy 2010). In 10-15% of patients' acute LBP develops into CLBP (Cilliers and Maart, 2013). This is characterized as pain lasting for more than three months (Liddle, Baxter and Gracey, 2004).

A recent systematic review of 13 international articles states that there is almost no agreement among researchers regarding the definition of LBP, where studies included various kinds of LBP (Fatoye. F., Gebrye, T., & Odeyemi, I., 2019). The various kinds included were acute, subacute, chronic, with radiating pain and without radiating pain (Fatoye. F., Gebrye, T., & Odeyemi, I., 2019). Annually, 15-20% of adults will report an incident of LBP, with 50% -80% reporting at least one episode of LBP throughout their lifespan (Deyo, Rainville and Kent, 1992; Dagenais, Caro and Haldeman, 2008). International epidemiological studies indicate lifetime prevalence rates of 70 -85% (Rubin, 2007; Hoy et al., 2010). A systematic review evaluating the prevalence of LBP internationally from 1966-1998 found that, prevalence varied widely (Walker, 2000). This was attributed to disparate methods of data collection, inadequate sample size, and cohort variability. Cohort variability included the non-standardisation of age and psychosocial function among different cohorts, varied levels of PA and inconsistent measurement and reporting of physical features (e.g., body mass index [BMI], gender, lumbar mobility, trunk strength and radiographic abnormalities) and general health status.

From the 56 studies included in a systematic review of international prevalence of LBP, although 11 of the studies were conducted in developing countries, none were conducted on African populations (Walker, 2000). This highlights a paucity of data on the prevalence of CLBP, in Africa. A systematic review and meta-analysis of 65 epidemiological studies on the prevalence of LBP in Africa describes a pooled lifetime, annual and point prevalence of LBP in Africa to be 47%, 57% and 39% respectively (Morris et al., 2018). Following Nigeria, the majority (25%, n=16) of these studies were conducted in South Africa (Morris et al., 2018). In 2016, data was obtained from a face-to-face survey of 10,336 adults in the 2016 South Africa Demographic and Household Survey (Kamerman et al., 2020). The prevalence of chronic pain was 18.3% (95% confidence interval [CI]: 17.0-19.7). Following limb pain, the second most frequent complaint of chronic pain was CLBP (30.5% [95%

CI: 27.7-33.6]). In summary, although there are not many studies, the prevalence of LBP in Africa appears to be higher or comparable to global LBP prevalence (Morris et al., 2018).

CLBP negatively affects patient's function and quality of life and can increase disability (Chou, 2010; Meucci, Fassa and Faria, 2015). Evaluations of the disability caused by CLBP are heterogeneous, with at least 26 different methods of evaluations being available in clinical practice (Longo et al., 2010). Several contributing categories to disability are affected by CLBP, and include pain intensity, personal care, lifting, walking, sitting, standing, sleep, sexual function, social activity and travelling (Fairbank and Pynsent, 2000). As LBP persists to CLBP, increased reports of disability and work absenteeism are noted (Woolf and Pfelger, 2003). This is however controversial due to the greater awareness of minor symptoms and increased willingness to report them thereby increasing the association with disability (Woolf and Pfelger, 2003). Such disability has economic implications for governments, businesses, communities, families, and individuals (Schwellnus et al., 2011). These economic implications and costs have been described as direct (medical expenses), and indirect (absenteeism, decreased productivity) (Dagenais, Caro and Haldeman, 2008).

2.2 Risk Factors

Risk factors implicated in CLBP in the aetiology of LBP internationally, have been broadly defined as demographic, health, occupational, psychological, and spinal anatomical factors (Table 1) (Rubin, 2007). This work concurs with work conducted in South Africa (Schwellnus et al., 2011), where sixteen risk factors were identified. However, as many as 55 different risk factors related to LBP have been identified (Hildebrand, 1987).

Table 1: Risk factors associated with CLBP

Risk factors according to Rubin, 2007.	Risk factors according to Schwellnus et al., 2011.
Socioeconomic status and education level	Low socioeconomic status and level of education
Age	Increased age
Gender	Female gender
Demographic factors	Drug abuse
Health factors	Psychosocial factors: stress, depression, work-related factors such as dissatisfaction and monotonous work
Monotonous tasks	
Job dissatisfaction	
Psychological factors including depression	
Body mass index (BMI)	Obesity (BMI>30/kg/m²)
Tobacco use	Smoking
Perceived general health status	Low self-rated health status
Occupational factors	Increased high risk physical activities (occupational& leisure time)
Physical activity, such as bending, lifting, or twisting	Heavy physical work, prolonged static work postures, heavy lifting, twisting, vibration
	Physical inactivity
Spinal anatomy factors including anatomical variations	Spinal anatomical factors e.g. severe scoliosis, transitional vertebrae
	Osteoporosis
	Arthritis
Imaging abnormalities	Pregnancy
	History of headache

The paradigm of assessing risk factors has moved from a biomedical towards a biopsychosocial assessment (O'Sullivan, 2005). This more recent paradigm attempts to explain the pain experience through cognitive, emotional, and sensory realms; expressing the dynamic interplay between biological, psychological and social factors rather than through biological and physical factors alone (Gatchel et al., 2007; Meyer, 2007). A biomedical model supports risk factors for CLBP that have been associated with anatomical variations and abnormalities linked to anatomical lumbar spine structures (Table 2) (Jackson and Simpson, 2006; Rubin, 2007). However, the predictive value of imaging to define anatomical abnormalities causing the pain is poor (Borenstein et al., 2001; van Tulder et al., 2006b; Chou et al., 2007).

Table 2: Lumbar spine pain sources (Jackson and Simpson, 2006).

Lumbar-sacral spine anatomical structures
Nucleus pulposus
Annulus fibrosus
Facet joint
Muscles
Ligaments
Synovium
Somatosensory system/ nerve

Furthermore, psychosocial risk factors are mentioned as a category for CLBP risk factors (Schwellnus et al., 2011). The South African study does not describe the multiple psychosocial factors contributing to CLBP. Psychological risk factors associated with CLBP that are independent of other variables and include stress, distress, anxiety, and depression (Linton, 2000). However, psychosocial variables such as pessimistic attitude, passive coping, and fear-avoidance beliefs have more impact than biomedical factors on CLBP disability (Linton, 2000; Woolf and Pfelger, 2003). These findings concur with those found in a systematic review (Raymond et al., 2011) reporting 23 papers and examining 16 psychosocial risk factors. Of these, catastrophizing and kinesiophobia were independently linked with poor outcomes in CLBP (Vlaeyen et al., 1995; Picavet, Vlaeyen and Schouten, 2002; Barke et al., 2016). Catastrophizing is defined as an exaggerated negative mental set brought to bear during actual or anticipated painful experience (Picavet, Vlaeyen and Schouten, 2002; Sullivan et al., 2009). Kinesiophobia is defined as an irrational and debilitating fear of physical movement and activity resulting from a feeling of vulnerability to painful injury or re-injury (Kori, Miller and Todd, 1990). In recognizing the biopsychosocial model, the significant predictive values of the risk factors, kinesiophobia and catastrophizing, are acknowledged (Barke et al., 2016, National Institute for Health and Care Excellence, 2016).

Over-reliance on risk factors for determining predisposition to CLBP can be problematic since risk factors vary with LBP definitions. Reliance on mostly self-reported or mechanical factors may explain inconsistencies between reviews. Related to different risk factors, in studies related to CLBP epidemiology, small sample size often limits their usefulness and reliability (Rubin, 2007). Further challenges in reliability include self-perceived health status often reported in epidemiological studies of participating CLBP patients (Raymond et al., 2011).

2.3 Lumbar spine anatomy implicated in chronic lower back pain

Structural abnormalities have been a historical focus in the literature (Figure 1 and 2; Table 2) (Jackson and Simpson, 2006; Vela, Haladay and Denegar, 2011). Figure 2 demonstrates the proximity of the nervous system to the articulations of the lumbar vertebrae. Implicated in biomedical models for classifying CLBP, anatomical structures of the lower back can lead to the development of CLBP (Langevin and Sherman, 2007; Allegri et al., 2016).

Figure 1: Lumbar vertebrae and disc including a lateral nucleus pulposus herniation

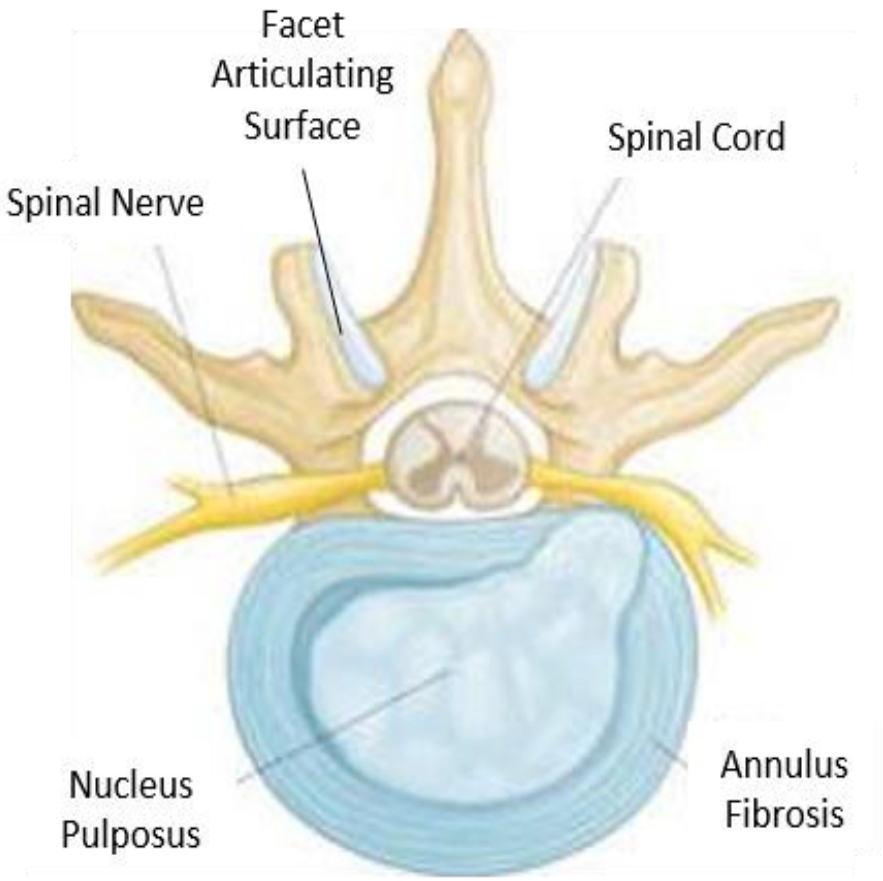

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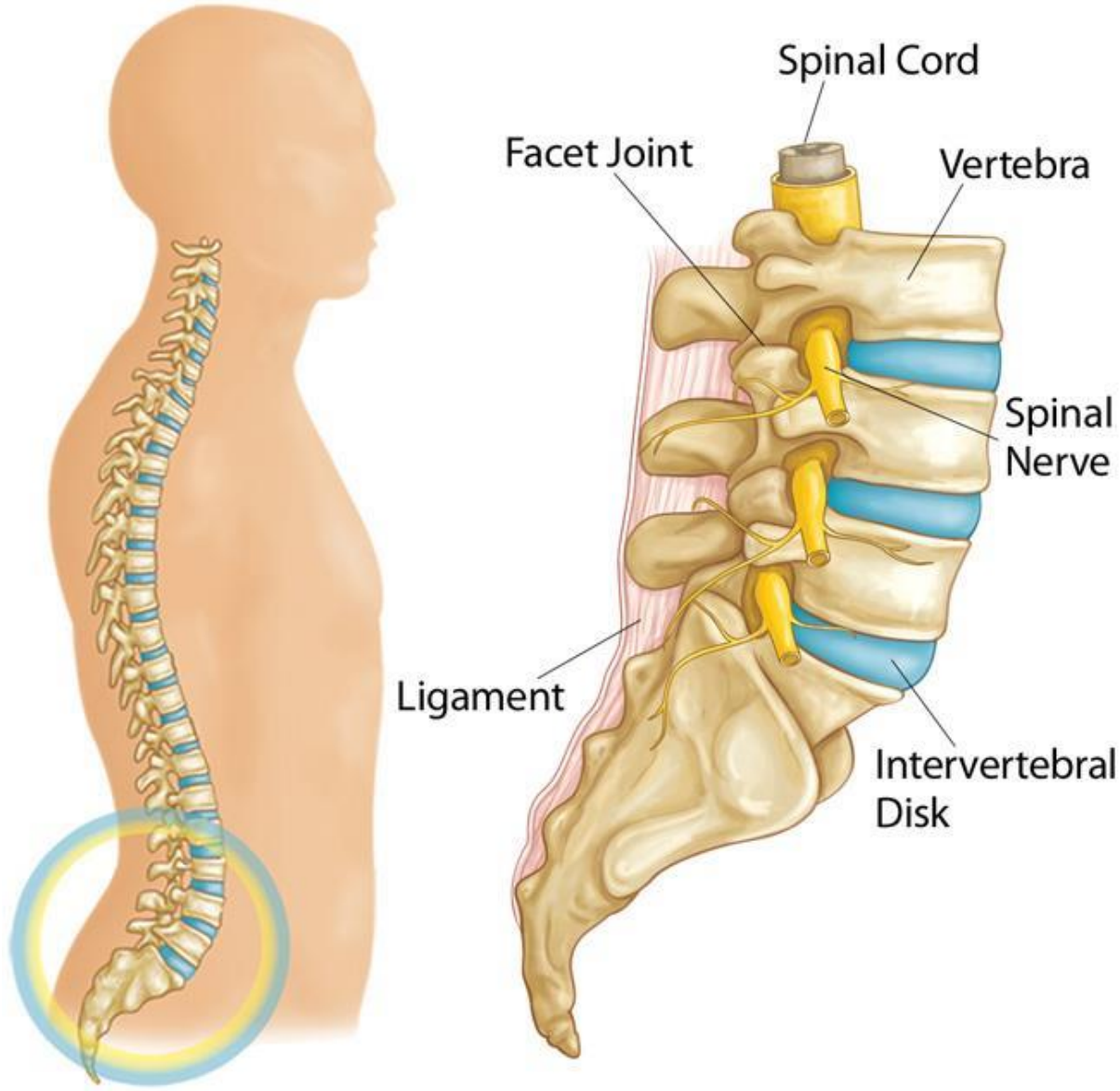
Figure 2: Lumbo-sacral spine



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Objectively verifying pathology in the lumbosacral structures has relied on diagnostic imaging by MRIs, CT scans and X-Rays. In contrast to the findings by Yang et al., (2015), where there was a positive correlation in symptoms of lumber disc degeneration and increased inflammatory factors with MRI findings (n=57), other studies have found that relying on imaging is not predictive of pain, with anatomical abnormalities also seen in healthy controls (Deyo and Weinstein, 2001; van Tulder, 2006b; Steffens et al., 2014; Allegri et al., 2016). In 85% of people pain is not attributable to visible pathology or intrusion into neural structures (van Tulder, 2006b). A systematic review of 12 LBP MRI studies, concluded poor LBP prognostic value associated to MRI patho-anatomical evidence

(Steffens et al., 2014). However, study limitations included a small sample sizes, heterogeneity of MRI findings and limited clinical outcomes between participants. Authors concur, symptoms, pathology, and radiological evidence remain poorly correlated to give specific diagnoses (Borenstein et al., 2001; van Tulder et al., 2006c; Chou, 2010; Steffens et al., 2014).

Additionally, concurring views acknowledged that leg pain can be associated with CLBP (Jackson and Simpson, 2006; Brukner and Kahn, 2012). Referred lower limb pain which originates in the lower back is said to be somatic or radicular in origin and can occur with or without LBP (Brukner and Kahn, 2012). Clinical history and physical examination can provide clues to which anatomical structure may have led to the origin of the pain but are not specific enough in describing which micro anatomical features and processes may be responsible for the pain (Dankaerts et al., 2006; Jackson and Simpson, 2006). Explanations of CLBP, focussing only on the lumbar spine, appear limited. Consideration of somatosensory system and neurophysiological processes which influence the pain experience may be necessary (Woolf, 2004; Davis et al., 2017).

2.4 Basic neurobiology of nociception and pain

Due to the clinical complaint of pain, it is salient that pain itself be examined prior to treatment.

Nociception

The sensory system responsible for the conscious perception of pain as well as pressure, touch, position, movement and vibration from the joints, muscles, fascia, and skin is called the somatosensory system. Nociception is not pain (Moseley, 2007). Nociception is sensation provoked by noxious mechanical, chemical, or thermal stimuli and is elicited as an early-warning physiological protection mechanism (Woolf, 2010). Nociceptive receptors of the somatosensory system are located in almost all body tissues (Dubin and Patapoutian, 2010). The framework supported in this trial outlines that pain is an indication of the complex experience and conviction of one's brain to protect body tissues and not as a marker of tissue damage (Moseley, 2007; Sullivan and Adams, 2010). A fundamental function of the somatosensory system is to signal actual or potential tissue damage, promoting withdrawal from the stimulus (Woolf, 2010).

Reference has traditionally been made to specific 'pain receptors' (Osterweis, Kleinman and Mechanic, 1987). However, more recently a distinction has been drawn between pain and nociception (Dubin and Patapoutian, 2010). Nociceptors are high threshold mechano and chemoreceptors. Acute injury sets off a complex cascade of chemical and electrical changes which sensitizes nociceptors depending on the magnitude and rate of activation (Purves et al., 2001). Damaged structures result in release of pro-inflammatory chemical irritants which activate peripheral

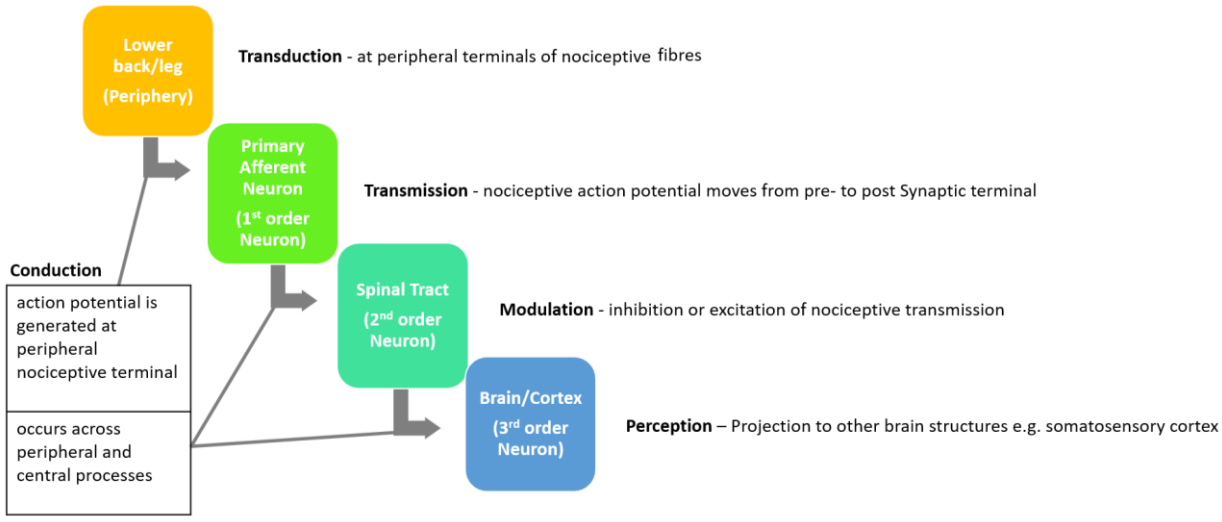
nociceptors causing depolarization of cell membranes. Nociceptive impulses are transmitted in A-delta and C fibres (Woolf, 2000; Koltzenburg, 1999). Nociceptor axons may be lightly myelinated A-delta fibres or unmyelinated C fibres. A-delta fibres have faster conduction velocities than C fibres. Both types of fibre transmit localized sensations which may be perceived as painful (Table 3) (Meyer et al., 2006; Dubin and Patapoutian, 2010). Nociceptive sensitization mechanisms may further explain the experience of pain. It is widely debated the interactive role of peripheral sensitization and central sensitization in chronic pain and CLBP.

Table 3: Nociceptive fibre myelination and impulse velocity. (Siegel and Sapru, 2011)

Nociceptive fibres	Conduction velocity	Impulse conduction velocity	Axon diameter	Myelination
A Delta	3-30 m/s	Fast	1-5 µm	Myelinated
C	0.5-2 m/s	Slow	0.2-1.5 µm	Unmyelinated

Nociception is relayed in phases through the somatosensory system, via 1st, 2nd, and 3rd order neurons. This is known as transduction, conduction, transmission, modulation, and perception (Figure 3) (Woolf and Ma, 2007). Saliently, impulse facilitation or inhibition is referred to as modulation (Staud 2013). The principal distinction between pain and nociception remains: pain emerges from the brain's evaluation of potential danger to the body (Moseley, 2007; Legrain et al., 2011; Sullivan, 2013).

Figure 3: Phases of nociception relayed through the somatosensory system.



Adapted from Brodal 1981

Pain pathways: 1st; 2nd and 3rd order neurons

Sensations from the lower back and leg delivered to the sensory cortex in the parietal lobe, arise via a three-neuron system beginning at the periphery, and ascend through spinal cord, brainstem, and thalamic relay nuclei pathways (Figure 3). Early research showed this system comprises of first order, second order and third order neurons (Brodal, 1981), enabling the hypothesis that pain is not a binary switch highlighting all nociception to the conscious awareness of sensations in the cortex (Woolf, 2010). This somatosensory system can be described as modulatory circuits facilitating and inhibiting pain (Ossipov, Dussor and Porreca, 2010).

The peripheral nervous system includes first order neurons. The central nervous system encompasses second and third order neurons. Various sensory receptors are the first point where nociception is relayed to the somatosensory system (Table 4). Sensory receptors corresponding with specific somatosensory axons, function by determining changes in their surrounding environment, varying in size, myelination, anatomical structure, and function (Bhatnagar, 2002; Dubin and Patapoutian, 2010). Dorsal root ganglia in the spinal cord housing the first order neurons, receive impulses from sensory afferents (Dubin and Patapoutian, 2010).

Table 4: Sensory receptor microanatomy (Parent, 1996; Bhatnagar, 2002)

Receptor Microanatomy
Expanded tip endings
Encapsulated nerve endings
Free nerve endings

Second order neurons relay nociceptive signals within the spinal cord. These neurons relay to the medulla oblongata, pons and midbrain, and end at the thalamus (Parent, 1996; Diaz and Morales, 2016). Excitation and inhibition mechanisms can alter transmission and function of second order neurons. Under ‘normal’ conditions, descending modulation to decrease painful sensations occurs at the midbrain (Periaqueductal gray matter); Pons (Locus coruleus); and Medulla (nucleus raphe magnus) (Dubin and Patapoutian, 2010). A review by D’Mello and Dickenson (2008) states that in neuropathic or inflammatory conditions, shifts can occur, heightening dorsal neurons response to afferent signals and increased signalling to the brain.

From the thalamus, third order neurons transmit information to cortical sensory areas forming a complicated functional network (Parent, 1996). The function of the primary somatosensory cortex is sensory information integration and determination of pain. Nociception is therefore the detection and transmission of information about potentially painful stimuli by nociceptors. Pain is a product of

higher brain centres. This network of synergistic connections in the higher centres allows for detection, attention, orientation, and reaction to actual or potential threats (Legrain et al., 2011).

The 'relay' stations of nociceptive pathways highlight that the experience of pain can be upregulated or downregulated at various levels affecting the pain state (Ossipov, Dussor and Porreca, 2010). Both 'bottom–up' and 'top–down' modulatory circuits within the spinal cord and brain are known to regulate the pain experience. Pain experiences among individuals are highly variable, corroborated by multiple studies (Dubin and Patapoutian, 2010; Staud 2013). This is attributed to both variable central nervous system processing and variation of sensitivities in peripheral pain receptors. Pain can be facilitated or inhibited by peripheral impulse modification in the dorsal horn. Additionally, central modulatory mechanisms have a regulatory effect. (Dubin and Patapoutian, 2010; Staud 2013). However, in an IASP consensus statement, the brain (through brain imaging) and its complex network, is now widely considered as the area where diagnosis, prognosis and treatment should be examined (Davis et al., 2017).

Pain

Eccleston and Crombez (1999) stated the qualities of the pain experience include pain being an unpleasant emotional experience, having unique perceptual and sensory characteristics, and no definitive correspondence between tissue damage and pain.

A review which defined and classified pain, maintains that pain is subjective (Kumar and Elavarasi, 2016). A prerequisite for effective pain management is accurate assessment of pain intensity (Charette and Ferrell, 2007). Pain intensity is typically measured by specific questionnaires such as the Numerical rating scale (NRS) and Visual Analogue Scale (VAS), which are used in physiotherapy and CLBP studies (Heymans et al., 2006; Kääpä et al., 2006). Concurring evidence states that ≥2 points on the 0–10 NRS or a change on the NRS of 20% between two time-points isrecognized as a minimal clinically important difference (MCID) (Haefeli and Elfering, 2006; Suzuki et al., 2020). A MCID on a VAS in CLBP is around 20mm, or 20% (Hägg, Fritzell and Nordwall, 2003; Ostelo and de Vet, 2005). Recognizing this, either can be used to in pain studies. Most tools to measure pain intensity have been developed in developed countries in Europe and America (Bagwath Perstad, Kamerman and Wadley, 2017). Recently, recognition of the NRS was established in African studies. The NRS worked well as a pain intensity outcome measure in an English speaking, educated cohort of SA university students in a model of experimental pain (Bagwath Persad, Kamerman and Wadley 2017). The NRS is recommended as a measure of change in pain intensity by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials

(IMMPACT) recommendations (Dworkin et al., 2008). However, it is also possible to detect pain objectively using fMRI imaging technology (Kucyi and Davis, 2015).

Acute pain is defined as pain that subsides within three months (Taxonomy, I.A.S.P., 2017). Pain involves a sophisticated interaction of immune, endocrine, and nervous systems (Chapman, Tuckett and Song, 2008). Physiological changes occur in the peripheral somatosensory system, spinal cord and brain in pain (Voscopoulos and Lema, 2010; Feizerfan and Sheh, 2014). Physiological studies show these changes differ in acute versus chronic pain (Voscopoulos and Lema, 2010; Feizerfan and Sheh, 2014).

Pain mechanisms

The function of the somatosensory system, and its contribution to the sensation of pain, is dynamic.

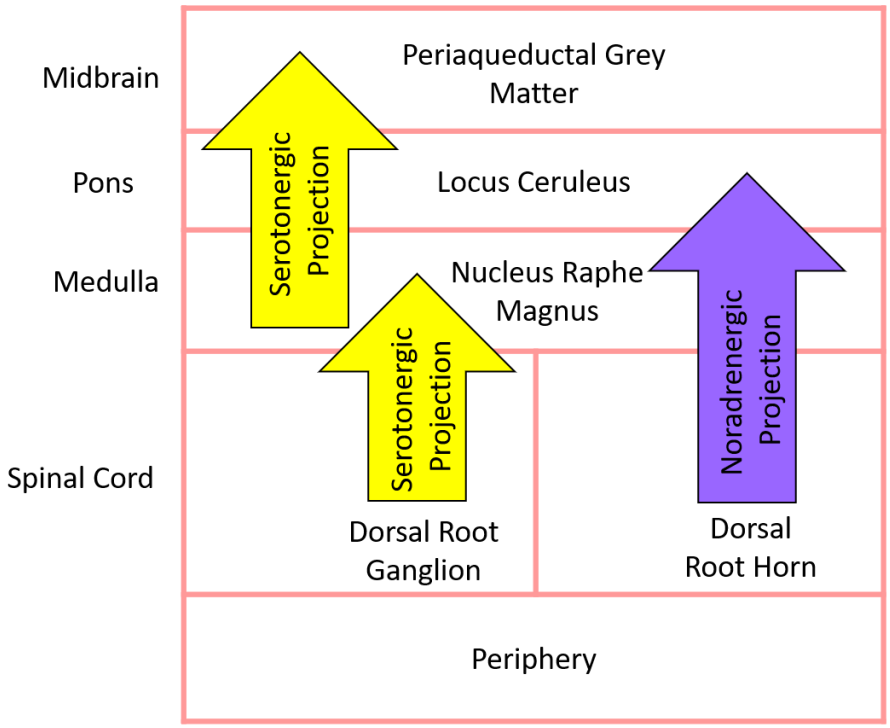
Peripheral sensitization: When peripheral sensory nerve fibres establish a reduced sensory threshold and when their response is magnified, this constitutes peripheral sensitization (Dubin and Patapoutian, 2010; Courtney, Fernández-de-Las-Peñas and Bond, 2017). In the dorsal root ganglia, nociceptive and non-nociceptive sensory afferents can become sensitized in chronic pain states (Gangadharan and Kuner, 2013). This sensitization can be due to an accentuated inflammatory response stimulating C fibres, inciting retrograde neurogenic inflammation (Meyer et al., 2006). Authors concur that the heightened sensitivity may result in more than expected pain from a painful stimulus (hyperalgesia) and pain perception from innocuous non-painful stimuli and normal body sensations (allodynia) (Julius and Basbaum, 2001; Costigan, Scholz and Woolf, 2009; Schweizerhof et al., 2009; Gangadharan and Kuner, 2013).

Central sensitization: Chronic pain also involves a mechanism of central sensitization whereby nociceptive neurons in the central nervous system have an increased responsiveness to noxious stimuli mediated at spinal and supraspinal levels (Woolf, 2011; Courtney, Fernández-de-Las-Peñas and Bond, 2017). A review describes central sensitization as a prolonged, however reversible phenomenon (Fleming and Volcheck, 2015). Central sensitization has been described as a CLBP phenotype existing alongside neuropathic and nociceptive pain (Smart et al., 2012a; Roussel et al., 2013). However, the co-morbidity of central sensitization with pain conditions including nociceptive pain (NP) and peripheral neuropathic pain (PNP) can be expected (Campbell and Meyer, 2006; Costigan, Scholz and Woolf, 2009; Woolf, 2011).

Understanding the involvement of somatosensory system and the brain in particular, with regards to pain, is now better understood. Modulation can occur at this first order neuron from regulation of inflammatory processes and conduction to the second order neurons, as well as interneuron activity

at the synaptic junction and descending pathways from the brainstem and cortex along the second order neuron (Figure 4) (Dubin and Patapoutian, 2010). With studies attempting to examine these signals further, heterogeneity is noted between the periphery and the brain. Nerve impulses in PNP phenotypes travel a more complicated path in the somatosensory system than with NP phenotypes (D'Mello and Dickenson, 2008; Garcia-Larrea and Peyron, 2013). This can be corroborated clinically, with delayed outcomes in PNP phenotypes (Chetty et al, 2012). A number of reviews highlight ascending and descending somatosensory system pathways, and multiple brain areas, that assist modulation of the experience of pain (Ossipov, Dussor and Porreca, 2010; Garcia-Larrea and Peyron, 2013; Courtney, Fernández-de-Las-Peñas and Bond, 2017).

Figure 4: Pain modulatory areas within the second order neuron.



Adapted from Dubin and Patapoutian 2010

The role of the brain in pain

Reviews have concluded that CLBP can affect brain morphology, chemistry, and neuronal activity (Ossipov, Dussor and Porreca, 2010; Garcia-Larrea and Peyron, 2013). Gray matter density is decreased in those with CLBP (Apkarian et al., 2004; Kregel et al., 2015). Compared to healthy controls, brain chemistry is altered in the thalamus, anterior cingulate cortex, and prefrontal cortex in chronic pain patients (Grachev, Fredrickson and Apkarian, 2000; Apkarian et al., 2004; Siddall et al., 2006). Unsuccessful pain modulation may be the result of a reorganized somatosensory cortex (a brain area associated with pain processing) and neuronal activity which is altered in people with CLBP (Flor et al., 1997; Giesecke et al., 2004; Diers et al., 2007; Lloyd et al., 2008; Kregel et al.,

2015). Recent fMRI objective evidence shows cortical changes take place in human chronic pain (Davis et al., 2017). Imaging techniques have shown that pain is associated with brain modulatory pathways, network, and region abnormalities. Spinal cord 'bottom-up' pathways activated by noxious stimuli, together with 'top-down' pathways which modulate ascending nociceptive signals operate in dysfunctional regulation. In people with chronic pain, the Salience network (encompassing mid-cingulate cotex, temporoparietal junction and anterior insula), sensorimotor network (encompassing the thalamus, primary somatosensory cortex, periaqueductal grey and posterior insula) and the default mode network (encompassing the posterior cingulate cortex and medial prefrontal cortex) activities are found to be abnormal (Davis et al., 2017).

More recently, further brain complexity has been seen where brain networks communicate intrinsically and dynamically and attentional states are persistently fluctuating (Kucyi and Davis, 2015; Davis et al., 2017). These brain regions involved in pain have been referred to as the pain matrix (Ossipov, Dussor and Porreca, 2010; Garcia-Larrea and Peyron, 2013). However, the term pain matrix has been superseded, since it refers to brain regions not necessarily specific to pain (Davis et al., 2017). The complexity of the somatosensory system interactions is recognized. The most recent model is termed the dynamic pain connectome (Kucyi and Davis, 2015; Davis et al., 2017). No single definition of boundaries of these interconnected networks exists, however there is some overlap in intrinsic salience, somatosensory and default mode networks from the previous pain matrix (Davis et al., 2017).

Abnormalities are noticed in brains of patients with CLBP (Davis, 2006). Conclusions were made highlighting abnormal forebrain responses to somatic stimuli implicating increased sensitivity in cortical somatosensory processing, abnormal attentional processes, poor endogenous pain modulation and atypical connectivity in normally connected cortical and subcortical networks (Davis, 2006). Authors concur that amplified somatosensory abnormalities exist in neuropathic pain (Davis, 2006; Gustin et al., 2011; Garcia-Larrea and Peyron 2013). This highlights the salience of pain phenotypes. Neuropathic pain has been defined as pain attributable to a lesion /dysfunction of the peripheral nerve, dorsal root ganglion or dorsal root (Woolf, 2004; Devor, 2006). This is unlike nociceptive pain that arises from actual or threatened damage to non-neural structures and is due to activation of nociceptors (Taxonomy, I. A. S. P., 2019). Acknowledging pain phenotype existence in CLBP, highlights the importance of the brain as a source of future investigation.

These multidimensional and not exclusively somatosensory operations highlight the clinical context, where pain has been described with sensory-discriminative and affective-motivational dimensions (Davis, 2006; Senkowski, Hofle and Andreas, 2014). Anatomically the former is associated with the primary and secondary somatosensory cortices, and the latter, the anterior cingulate cortex, insular

and prefrontal cortex (Senkowski and Heinz, 2016). The affective-motivational dimension is related to psychological, motivational, and higher cognition (empathy and relative demands to pain) and reflects perceived unpleasantness of the pain experience. The sensory discriminative dimension fulfils the perceived intensity, quality, and location of pain. This according to Davis et al., (2017) corroborates the limitations of the dynamic pain connectome in chronic pain due to brain activity varying across time, people, and context.

Limitations of neuroimaging techniques include limitations in associated statistical modelling, the impact of testing conditions, understanding of normal ranges within individual noxious stimuli and population's variance in pain evocation (Davis, 2006). In context, an IASP presidential task force in 2015 examined the capabilities of brain imaging to diagnose chronic pain as having ethical, economic, and legal implications (Davis et al., 2017). A final statement was made stating imaging cannot exclusively define an individual's chronic pain (Davis et al., 2017). However, neuroimaging evidence brings to light the need to consider the neurophysiological alongside biopsychosocial models when examining CLBP (Davis, 2006; Ossipov, Dussor and Porreca, 2010; Senkowski and Heinz, 2016). The limitations of objective measures and lack of clinical utility elucidates the need for more information when classifying and diagnosing CLBP.

2.5 Clinical decision making

Classification of chronic lower back pain

Anatomical or pathological aetiology cannot be used to classify CLBP in 90% of cases (Koes, van Tulder and Thomas, 2006; Spahr et al., 2017). Since imaging is not a reliable indicator of pathology, the underlying pain mechanism cannot be assumed (O'Sullivan, 2005). Patients often present with symptoms with no clear, specific cause, and are classified as having non-specific lower back pain (NSLBP) (O'Sullivan, 2005; Dankaerts et al., 2006; Schwellnus et al., 2011). Specific lower back pain involves an inclusion of a specific pathology, e.g.: Cauda equina syndrome, fracture (Koes, van Tulder and Thomas, 2006). The classification of specific and non-specific CLBP has limitations, due to multiple factors contributing to the classification (O' Sullivan, 2005). Clinically useful classifications should be based on mechanisms driving the disorder; this in turn may predict the outcome (O'Sullivan, 2005). Several models including the pathoanatomical, neurophysiological, psychosocial, peripheral pain generator, mechanical loading, signs and symptoms, motor control and biopsychosocial models, have been used for classification and diagnosis of CLBP (O'Sullivan, 2005).

Pathoanatomical, mechanical loading and signs and symptoms models are similar. They are based on assumptions that CLBP interventions should be aimed at structural abnormalities like

intervertebral disc degeneration (including annulus prolapse and tears), facet-joint degeneration, spondylolisthesis, spinal stenosis, with or without nerve pain (Borenstein and Calin, 2012). Limitations of the pathoanatomical model include evidence that these abnormalities are found in pain free individuals and little attention is paid to neurophysiological and psychosocial variables (Nachemson, 1999; Humphreys and Irgens, 2002; Dankaerts et al., 2009).

The mechanical loading model has several causative factors associated with CLBP symptoms. These include very high and low levels of lumbar spine loading, repeated or heavy lifting in flexion, sustained positions at awkward angles, frequent or intense loads in sport or at work, increased BMI, joint hypermobility, limited trunk strength, limited lower limb flexibility and overloaded spinal segments out of their normal ranges of movement (Balague, Troussier and Salimen, 1999; Heneweer et al., 2011; Trompeter, Fett and Platen, 2017). Similarly, neurophysiological, and psychosocial contributors are sparsely accounted for.

Based on pathoanatomical and biomechanical assessment, the signs and symptoms model is usually supported by physiotherapists internationally and in South Africa, to classify CLBP (Maitland, 1986; Delitto, Erhard and Bowling, 1995; McKenzie, 2000; Naidoo et al., 2012). This model relies on area and nature of pain symptoms, functional and motor control changes, vertebrae mobility and sensitivity to mechanical provocation, and centralization or peripheralization of the pain (Maitland, 1986; McKenzie, 2000). However, consideration of biopsychosocial factors is limited.

Similarly, the peripheral pain generator model relies on identifying the painful structure, through clinical examination, patient history, and diagnostic blocks using intramuscular and intra-articular zygapophysial joint anaesthetic injections (Bogduck, 1995). Treatment incorporates identification of the painful structure and blocking or denervating the nociceptive source (Carette et al., 1991; O'Sullivan, 2005). This model also does not account for psychosocial components (Carette et al., 1991; Nachemson, 1999).

The motor control model claims mal-adaptive movement and motor control occur with CLBP (O'Sullivan, 2005). It was reliant on mal-adaptive movement driving peripheral nociception (O'Sullivan, Twomey and Allison, 1997). Problems with this model included model-derived treatment aimed at muscular control when additional variables from a psychological or neurobiological driver may have caused the motor control problem (Hodges and Moseley, 2003). To mitigate these limitations, the model has evolved to include three subgroups: adaptive altered motor responses to underlying disorders, altered motor responses secondary to psychological factors and mal-adaptive motor problems driving CLBP (O'Sullivan, 2005).

In the neurophysiological model, the focus is on neuromodulation and biochemical changes in the somatosensory system (Freynhagen et al., 2006; Smart et al., 2012a; Spahr et al., 2017). A mechanism-based classification is used for CLBP where pain is classified by the dominant neurophysiological mechanism responsible for its origin and/or maintenance in order to treat pain phenotypes effectively (Smart et al., 2012a). Differentiation of nociceptive, neuropathic, and central sensitization CLBP phenotypes are used within this model but remain controversial. This controversy is due to differences between definitions and diagnostic outcomes (Smart et al., 2012b; Spahr et al., 2017). For example, the definition of neuropathic pain involves a demonstrable lesion of the somatosensory system (Taxonomy, I.A.S.P., 2017). However, contradictory clinical evidence shows many patients without demonstrable lesions present with symptoms indicating neuropathic pain (spread of pain, allodynia, dysesthesia) (Spahr et al., 2017).

The psychosocial model implicates social and psychological factors in driving the somatosensory system to modulate pain (Zusman, 2002; Waddell, 2004). The broad spectrum of social factors includes financial compensation, home and work tensions, and cultural beliefs; all of which can affect pain. Both disability and pain can be affected by psychological variables for example catastrophizing, pathological fear and anxiety, avoidant behaviour, hyper-vigilance, and negative thinking, which are mal-adaptive (Nachemson, 1999). Despite the debate as to which of these factors predispose the pain, authors concur maladaptive coping can be reduced through descending inhibition via distraction, appropriate pacing, and adaptive coping measures (O'Sullivan, 2005; Peters, Vlaeyen and Weber, 2005). The argument exists that there is an over reliance by physiotherapists to implicate psychosocial factors in Non-Specific Chronic Lower Back Pain (NSCLBP) with only a small group having this as the primary driver for the disorder (O'Sullivan, 2005).

Due to its multidimensional approach, the biopsychosocial model is widely accepted and used by physiotherapists treating CLBP (Turk and Gatchell, 2002; O'Sullivan, 2005). Due to the multifactorial nature of this model, the driver for pain is determined using clinical expertise, and the relevant information gained through interviews, clinical neuromuscular assessment, and perhaps radiological screening (O'Sullivan 2005; Waddell, 2004). Previously biomedical models were emphasized, however, a systematic review of 49 papers highlighted that social factors are often overlooked in their contribution to CLBP (Froud et al., 2014). The biopsychosocial model, however, has been criticised for its epistemological underpinnings and misuse, because it contains no safeguards against over/ under representation of biological, psychological, or social domains (Benning, 2015).

To improve treatment outcomes, subgroups of CLBP have been identified (Delitto, 2005; Stanton and Kawchuk, 2008; Huijnen, 2015). A review examining physiotherapy interventions based on

subgroup classification (Alrwaily et al., 2016) concurs with the potential to enhance effect sizes in studies with identical CLBP interventions if participants are classified by type of CLBP (O' Sullivan, 2005; Delitto, 2005; Dankaerts et al., 2006; Stanton and Kawchuk, 2008; Huijnen, 2015).

In two recent reviews, physiotherapy treatments were matched according to subgroup classification (Karayannis, Jull and Hodges, 2012; Alrwaily et al., 2016). For example, mechanical diagnosis was designed to identify non-musculoskeletal pain and emphasize self-treatment such as the McKenzie Method of exercise (McKenzie, 2000). In movement system impairment classification, treatment is based on correcting the impaired alignment and movement patterns as well as correcting the tissue adaptations associated with the impaired alignment and movement patterns using manipulation, massage and stretching as part of therapy (Sahrmann et al., 2017). The majority of studies reviewed relied on biomechanical assessment (Karayannis, Jull and Hodges, 2012). However, one classification developed by O'Sullivan (2005), considered neurophysiological and psychosocial aspects. In Alrwaily et al., (2016), four classification subgroups sparsely explored psychosocial factors. Both Ford and Hahne (2013) and Alrwaily et al., (2016) concur that no classification is completely comprehensive, since different CLBP clinical pictures account for variation during the treatment period, and some systems require specialist knowledge about treatments matching diagnostic classification. In absence of this knowledge, success of treatment by subgroup classification may be limited. Incorporation of valid psychosocial outcomes within treatment interventions should be encouraged with clinicians recognizing that variables such as kinesiophobia and pain catastrophizing may require management in CLBP cohorts. Elucidating subgroup classification according to neurophysiological phenotype may enhance statistical modelling when examining CLBP reducing the paucity of knowledge in pain nuerophysiology.

Neurophysiological classifications of CLBP have only recently been explored in physiotherapy assessment (Courtney, Fernández-de-Las-Peñas and Bond, 2017). This classification is not yet explicitly used in treatment guidelines. A study of 55/100 English respondents in chronic pain sufferers were treated as a homogenous group with standardized treatments (Brown, 2004). Albeit not only CLBP, pain phenotyping was not used for subgroup classification. A review by Woolf (2010) classifies pain as nociceptive pain, inflammatory pain, or pathological pain. Pathological pain can be argued as having two subtypes: neuropathic pain, and dysfunctional pain (Woolf, 2004; Costigan, Scholz and Woolf, 2009). Dysfunctional pain occurs with no identifiable noxious stimulus or apparent inflammation or damage to the somatosensory system. It is widespread or isolated, typically with hyperalgesic presentation (Costigan, Scholz and Woolf, 2009). Neuropathic pain typically presents with dysesthesia (abnormal unpleasant symptoms) and responses to noxious and innocuous stimuli are enhanced (Costigan, Scholz and Woolf, 2009). Each pain phenotype is driven by a different neurobiological process (Woolf, 2010). Early warning physiological protection and

guarding against noxious stimuli defines nociceptive pain (Costigan, Scholz and Woolf, 2009; Woolf, 2010). Inflammatory pain is a result of inflammation activating nociceptors, increasing peripheral sensory sensitivity (Costigan, Scholz and Woolf, 2009; Woolf, 2010). Acute inflammation is therefore functional, and chronic inflammation is not. Pathological pain is not a symptom of a disorder but is due to a diseased state of the somatosensory system (Woolf, 2010). The difference lies in pathological pain resulting from abnormal neural processing, whereas inflammatory pain occurs due to a demarcated peripheral pathology causing a hypersensitive reaction (Woolf, 2010).

Neurophysiological classification exhibits challenges. Experts concur that PNP is defined by spontaneous pain and hypersensitivity to pain with a lesion or damage of the somatosensory system (Woolf, 2004; Costigan, Scholz and Woolf 2009; Woolf, 2010). However, this statement is challenging. Even though abnormal neural plasticity may occur, and nociceptive signals may be enhanced in response to noxious and innocuous, not normally painful stimuli, a lesion is necessary but not always sufficient to generate neuropathic pain (Costigan, Scholz and Woolf, 2009). Concurring with this, Spahr et al., (2017), advocates that a significant neuropathic component to CLBP may be present, irrespective of any demonstrable lesion or pathological status. Now, in addition to nociceptive and neuropathic pain, a third mechanistic descriptor of pain has been created: nociplastic pain. Nociplastic pain arises from altered nociception, despite no clear evidence of actual or potential tissue damage resulting in peripheral nociceptor activation, or evidence for a lesion or disease of the somatosensory system (Kosek et al., 2018). What is agreed currently is that pain occurs in select phenotypes (Taxonomy, I. A. S. P., 2019). In summary, combining neurophysiological classifications and the biopsychosocial model may be warranted for physiotherapy diagnosis (Waddell, 2004; Elvey and O'Sullivan, 2004).

Diagnosis

There are multiple interacting factors that influence the development of CLBP (O'Sullivan, 2005). Previously described classification systems, which aid diagnosis are of finite clinical value unless the underlying mechanism(s) driving the pain disorder are identified (Wand and O'Connell, 2008). The biopsychosocial model is widely accepted as the most holistic approach to diagnosing and treating chronic pain (Gatchel et al., 2007). A recent paper encourages physiotherapists to identify pain according to neurophysiological drivers (Courtney, Fernández-de-Las-Peñas and Bond, 2017). However, CLBP diagnosis continues to lack evidence linking neurophysiological drivers within this model when applied to patient diagnosis.

It is possible to assign a pain phenotype to CLBP according to neurobiological mechanisms (Freynhagen and Baron, 2009; Spahr et al., 2017). For example, nociceptive and neuropathic subgroupings have been used for some time to base individual pain diagnosis (Smart et al., 2012b;

von Hehn et al., 2013, Spahr et al., 2017). Using pain phenotype subgroupings for diagnostic purposes however has not been explored in physiotherapy and walking studies of CLBP patients.

Guidelines from the current International Association for the Study of Pain (IASP) distinguishing neuropathic CLBP from nociceptive CLBP rely on a demonstrable lesion or disease within the somatosensory system in neuropathic pain (Taxonomy, I.A.S.P., 2017) Many patients are misdiagnosed as not having a neuropathic component to their pain when it may be present (Freynhagen et al., 2006; Attal et al., 2010; Campbell et al., 2013; Spahr et al., 2017). In IMMPACT (Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials) recommendations for diagnostic purposes, self-report screening instruments should not replace clinical examination (Edwards et al., 2016). However, phenotyping CLBP can be aided using validated reliable diagnostic questionnaires such as the painDETECT (Bennet et al., 2007; Edwards et al., 2016; Spahr et al., 2017). The painDETECT questionnaire is endorsed diagnostically, demonstrating that patients may present with a significant neuropathic component to their pain irrespective of any demonstrable lesion or pathological status (Spahr et al., 2017). The paucity of physiotherapy knowledge regarding neurobiological processes of CLBP is documented (Naidoo et al., 2012; Smart et al., 2012b; Ouedraogo et al., 2012; Chetty et al., 2012). It may be relevant to encourage physiotherapists to use the painDETECT questionnaire to aid diagnosis.

Pain phenotypes involved in chronic lower back pain

CLBP is a heterogeneous disorder due to underlying pathophysiological mechanisms, including patients with dominant nociceptive and neuropathic pain phenotypes (Scholz et al., 2009; Walker and Williamson, 2009; Smart et al., 2012c; Förster et al, 2013; Nijs et al., 2015). Despite no unified hypothesis explaining pathophysiology of all pain states (Bridges, Thompson and Rice, 2001), the mechanism-based classification of pain is categorized according to the main neurophysiological mechanism involved through inception to maintenance of the pain state (Woolf et al., 1998). According to Cruccu et al., (2004), chronic pain can occur as five phenotypes of pain (Table 5). A recent advance by the IASP added a further mechanistic descriptor of pain, namely nociplastic pain, to cover cases not adequately covered by “nociceptive pain” or “neuropathic pain” (Kosek et al., 2017). This literature review will concentrate on nociceptive and neuropathic pain phenotypes.

Table 5: Chronic pain phenotypes (Cruccu et al., 2004).

Five types of chronic pain
Nociceptive
Neuropathic
Mixed
Psychogenic
Idiopathic

There are several pain phenotyping questionnaires in existence (Kaki, El-Yuski and Youseif, 2005; Bennet et al., 2007; Jay and Barkin, 2014; van Hecke et al., 2015). To differentiate nociceptive from neuropathic pain, two most frequently used are the painDETECT and the Leeds assessment of neuropathic symptoms and signs (LANSS) questionnaires (Kaki, El-Yuski and Youseif, 2005; Freynhagen et al., 2006; Bennet et al., 2007; Jay and Barkin, 2014; van Hecke et al., 2015). Two studies phenotyped CLBP patients into nociceptive and neuropathic pain subgroups using valid and reliable questionnaires (Hassan, Saleh and Baroudy, 2005; Spahr et al., 2017). One study with 100 patients in the Middle East used the LANSS questionnaire (Hassan, Saleh and Baroudy, 2005). Another study in London with 50 CLBP patients used the painDETECT questionnaire (Spahr et al., 2017). The painDETECT has a sensitivity of 84%, and a specificity of 84%, where the LANSS has sensitivity and specificity ranging from 82% to 91% and 80% to 94% respectively (Potter et al., 2003; Kaki, El-Yuski and Youseif, 2005). However, they are not a substitute for clinical examination (Baron et al., 2016; van Hecke et al., 2015).

A recent review demonstrated differences in the nature and consequences of pain, by phenotype; PNP was associated with greater pain intensity, disability and was more costly to treat (Baron et al., 2016). This neurophysiological viewpoint concurs with a paper reviewing pain phenotype mechanisms giving depth to patient presentations within chronic pain cohorts (Courtney, Fernández-de-Las-Peñas and Bond, 2017).

<u>Nociceptive lower back pain</u>

Nociceptive pain (NP) can arise from noxious stimulation of the lumbar spine or associated soft tissues (Costigan, Scholz and Woolf, 2009; Hoheisal et al., 2013). The IASP defines NP as “pain that arises from actual or threatened damage to non-neural structures and is due to activation of nociceptors” (Taxonomy, I. A. S. P., 2019). Referred somatic pain can occur in parallel with CLBP of nociceptive origin (Arendt-Nielson and Svenson, 2001). It is suggested that symptomatic features rather than clinical signs are more useful to assume dominance of NP (Smart et al., 2012a).

NP is brought about by a noxious stimulus which stimulates peripheral receptive terminals of Aδ and C fibres (Ekman and Koman, 2004; Julius and McCleskey, 2006). Authors concur LBP may arise due to chemical alterations (Butler and Matherson, 2000; McMahon, Bennet and Bevan, 2006). Soft tissue ischemia allowing a decrease in pH of surrounding tissues or following the inflammatory cascade inducing chemically mediated nociception (Butler and Matherson, 2000; McMahon, Bennet and Bevan, 2006). Both localized and referred pain is possible in NP. Localised CLBP of nociceptive origin symptoms arise from non-neural structures (injured vertebrae, ligaments and discs) provoking nociception, in turn stimulating the sensory discriminative function of the somatosensory cortex (Arendt-Nielson and Svenson, 2001; Bushnell and Akparin, 2006).

Helliwell et al., (2003) supported pain classification using an absence together with presence of symptoms and signs. To differentiate pain phenotypes, screening instruments phenotyping pain as nociceptive or largely neuropathic (having a neuropathic component) are listed in Table 6 (Bennet et al., 2007).

Table 6: Neuropathic screening instruments (Bennet et al., 2007)

Neuropathic screening instruments	References
painDETECT	Freynhagen et al., 2006
Standardised Evaluation of Pain	Scholz et al., 2009
Douleur Neuropathique 4	Bouhassira et al., 2005
Neuropathic Pain questionnaire	Krause and Backonja, 2003
Leeds Assessment of Neuropathic Symptoms and Signs	Bennet et al., 2007

Diagnosis involves varying intensities of unpleasant abnormal sensations known as dysesthetic symptoms (Table 7). Using a regression analysis to identify a cluster of predictive criteria, presence of dysesthesias decreased the odds of being classified with NP by 85% (Smart et al., 2012a). In a diagnosis by exclusion, the absence of nerve damage associated with NP may be predicted by the absence of dysesthesias (Smart et al., 2012b).

Table 7: Dysesthesia symptoms typical of neuropathic pain: qualities used for discrimination between nociceptive and neuropathic pain phenotypes (Bennet et al., 2007; Smart, O'Connell and Doody, 2008; Freynhagen and Baron, 2009; Smart et al., 2012b).

Dysesthesia symptoms of PNP in neuropathic questionnaires
Burning/hot sensations
Electrical shock quality/ shooting pain
Crawling/ pricking/tingling/ paroxysmal pain
Cold/ numbness
Heavy feeling
Dysesthesias and pain in a dermatomal distribution
Pain evoked by innocuous stimuli like light touch/ spontaneous pain

Qualities of pain may predict NP (Smart et al., 2012a; Smart et al., 2012b). When resting, pain may present as dull, aching or throbbing due to nociceptor stimulation by constant inflammation or initial nociception (Gifford and Butler, 1997). Mechanical stimulation of primary nociceptors and signal transmission by Aδ fibres can explain sharp and intermittent pain nature (Butler and Matherson, 2000; Smart et al., 2012a). Concurring evidence states movement away from the painful nociceptive stimulus may be for protection of structures due to increased fear, stress, and attention together with CNS motor planning changes associated with the anterior cingulate cortex (Hodges and Moseley, 2003; Hall and Elvey, 2004; Smart et al., 2012a).

Neuropathic lower back pain (peripheral)

Authors concur that when compared to NP, patients with CLBP of neuropathic origin have increased pain intensity and disability, greater psychological co-morbidities, lower health related quality of life, and increased medical expenses (Bouhassira et al., 2008; Freynhagen and Baron, 2009; Smith et al., 2012; Spahr et al., 2017). Four hundred chronic pain patients were assessed using the painDETECT questionnaire confirming the negative impact of PNP (Shaygan, Böger and Kröner-Herwig, 2013). Compared to NP, the patients with PNP symptoms had significantly higher levels of pain intensity, disability, higher levels of depression and pain chronicity together with longer hospital stays (Shaygan, Böger and Kröner-Herwig, 2013).

Peripheral Neuropathic Pain (PNP) based on a mechanisms-based classification of pain is distinguishable from NP and central sensitization (Bennet et al., 2007; Smart, O'Connell and Doody, 2008; Costigan, Scholz and Woolf, 2009). The definition of PNP is pain attributable to a lesion /dysfunction of the peripheral nerve, dorsal root ganglion or dorsal root (Woolf, 2004; Devor, 2006). CLBP with a neuropathic component may have no history or confirmatory evidence despite a contrary clinical picture (Spahr et al., 2017). Clinical evidence of patient's signs and symptoms allow

CLBP patients classification with a neuropathic component irrespective of any lesion or pathological status (Spahr et al., 2017). This contributes to the definition of maladaptive plasticity caused by a lesion and not solely a consequence of a disease process or lesion (Spahr et al., 2017). Characteristically responses to noxious and innocuous stimuli are enhanced (Costigan, Scholz and Woolf, 2009).

A narrative review suggested 16% to 55% of people with CLBP have a neuropathic component (Baron et al., 2016). CLBP contributes greatly to global PNP (Chetty et al., 2012). Fifty percent of black Africans (Ouedraogo et al., 2012), and 55% of adults seeking outpatient care for CLBP in the Arabian Gulf (El Sissi et al., 2010) may exhibit a neuropathic component. No estimates of South African neuropathic CLBP have been reported (Chetty et al., 2012).

Despite varied aetiologies of neuropathies, authors concur that in CLBP, these aetiologies occur through inflammation, ischemia, compression, or trauma (Bridges, Thompson and Rice, 2001; Woolf, 2004; Devor, 2006). Neuropathic CLBP can arise from mechanical compression of a nerve root, damaged nociceptive sprouts inside a degenerating disc and/or degenerative disc inflammatory mediators causing nerve root inflammation and damage (Baron et al., 2016).

Animal models have been developed to explain neuropathic pain physiology (Li et al., 2000; Bridges, Thompson and Rice, 2001; Hoheisel et al., 2013; Strong et al., 2013; Baron et al., 2016). However, they do not typify the human response to somatosensory system injury. Animals under experimental conditions cannot verbalize their experience unlike humans, which is the hallmark of human PNP physiotherapy diagnosis, by using phenotyping questionnaires. Several authors concur that notable changes are associated with PNP in the peripheral somatosensory system (Bridges, Thompson and Rice, 2001; Costigan, Scholz and Woolf, 2009; Smart et al., 2012b). Notable changes are mostly clinically observed as signs and symptoms including ectopic discharges and ephaptic conduction of nerve impulses. The former occurs when the firing threshold is reached in a primary afferent neuron without the input of a stimulus (Wall and Gutnick, 1974; Yoon, 1996; Li et al., 2000). The latter occurring when A-fibres and C-fibres seen to demonstrate sub-threshold membrane oscillations causing thresholds reached sooner, and neuronal cross excitation through their membranes (Amir, Michaelis, and Devor, 1999; Bridges, Thompson and Rice, 2001; Vascopoulus and Lema, 2010). Cross excitation can occur between A and C fibres (Amir and Devor, 2000).

CLBP of neuropathic origin can be localised or referred, including radicular pain and radiculopathy which are distinct from somatic referred pain seen in NP (Baron et al., 2016). Radiculopathy is an objective sensory and/ or motor functional loss due to nerve root damage. It can occur with or without pain (Baron et al., 2016). Alternate evidence shows CLBP of neuropathic origin is not restricted to

typical radicular presentations (Attal et al., 2011; Campbell et al., 2013). This explains why clinically, atypical presentations may lead to poor treatment execution (Spahr et al., 2017).

Diagnostically, imaging measurements are not used as a gold standard for PNP. However, inference is made on clinical symptoms and signs corresponding to PNP mechanisms (Finnerup and Jensen, 2006; Baron et al., 2016). Imaging demonstrates spinal nerve root compression in 4%-17% of patients without pain (Boos and Lander, 1996; Weishaupt et al., 1998). CNS modulation though descending inhibitory and facilitatory mechanisms may explain why injuries which could cause PNP do not in all people (Finnerup et al., 2007a; Finnerup et al., 2007b; Costigan, Scholz and Woolf, 2009).

Therefore, use is made of screening tools identify PNP qualitative symptoms which present in combinations or alone (Bennet et al., 2007). Symptoms typical of PNP are noted in Table 7. Differentiating neuropathic from nociceptive CLBP phenotypes is made possible by screening tools such as the painDETECT (Freynhagen et al., 2006; Bennet et al., 2007; Spahr et al., 2017). A timeous musculoskeletal examination, spinal palpation and assessment of the patients' motor, sensory and autonomic nervous systems would help identify the neurological dysfunction and structures responsible (Haanpää et al., 2011; Nijs et al., 2015). PNP symptoms are neither universally present nor constitute an absolute diagnosis. Their presence, however, increases the percentage of a PNP component (Freynhagen et al., 2006; Rehm, Koroschetz and Baron, 2008).

Typical positive signs are seen in both rat animal models and humans (Bridges, Thompson and Rice, 2001; Baron et al., 2016). These signs are largely due to ectopic activity, include spontaneous pain, pain-like behaviour (not stimulus induced), and hypersensitivities: allodynia and hyperalgesia (Bridges, Thompson and Rice, 2001; Baron et al., 2016). Painful positive signs can also be evoked (stimulus induced hypersensitivity) e.g., worsening of pain by benign slow repetitive noxious stimuli (Rehm, Koroschetz and Baron, 2008). Spontaneous pain can be burning, shooting and electric shock-like in nature (Rehm, Koroschetz and Baron, 2008). Abnormal sensations are frequently reported with maximal pain often extending over this area with some patients reporting hypersensitivity with no sensation impairment (Rehm, Koroschetz and Baron, 2008). Typical positive signs reported include non-painful paraesthesia's (tingling, crawling). Signs of PNP may be resultant from nerve root inflammation, compression, and nerve sprouts that pathologically innervate a spinal disc (Baron et al., 2016).

Available treatment guidelines advocate a multimodal approach with pharmacotherapy for symptom relief together with PA and behavioural interventions (Baron et al., 2016). Authors concur, improvements in therapies are still required (Finnerup et al., 2007; Courtney, Fernández-de-Las-

Peñas and Bond, 2017). Non-pharmacological treatments for CLBP of neuropathic origin include transcutaneous electrical nerve stimulation (TENS), spinal cord stimulation, epidural steroid injections (Kumar, Abbas and Rizvi, 2012; Morlion, 2013). Specialist physiotherapy including cognitive behavioural therapy is mentioned as treatment of CLBP with neuropathic origins (Baron et al., 2016). No explicit descriptions of specialist physiotherapy are available.

Mixed pain

Many clinical presentations appear to be a mix of nociceptive, central and peripheral neuropathic mechanisms although a dominance of one neurobiological mechanism may be apparent clinically (Bennet et al., 2007; Schäfer et al., 2009; Fishbain et al., 2014). According to Picelli et al., (2016), CLBP should be considered a mixed pain syndrome. The conference paper states that 50%-70% of LBP is identified as NP, where PNP is present in 5%-15% of cases (Picelli et al., 2016). The pain DETECT demonstrates dominance of a phenotype in CLBP with the screening result final score comprising of three scenarios: a PNP component being unlikely (15%), an ambiguous result however a PNP component may be present; or a likely PNP component present (90%) (Freynhagen et al., 2006).

Despite diagnosis of mixed pain, studies and clinical pictures continue to demonstrate a dominance of a pain phenotype. In Shaygan et al., (2013), 37% (148/400) of patients had a dominance of PNP. Using the painDETECT, 48% (24/50) of CLBP patients showed a dominance of PNP (Spahr et al., 2017). By understanding pain phenotypes, physiotherapists and patients alike may be educated into rationale why not all CLBP pain complaints follow homogenous trajectories. This may add value in education and psychological support of patients.

Psychological factors involved in the pain experience in physiotherapy

Gate control theory describes how pain can be reduced at the spinal cord level by another sensory input (Melzack and Wall 1965). However, pain can be affected at other interfaces outside of the spinal cord. Holistic assessment and treatment is required due the complex nature of CLBP. Pain is considered as a combination of factors namely sensory, emotional, cognitive-evaluative, interpersonal, and cultural. In a review of 18 articles of psychosocial factors and pain chronicity and 83% (15/18) reported an association of psychosocial factors and pain chronicity (Hruschak and Cochran, 2018). Psychosocial variables such as kinesiophobia and pain catastrophizing are implicated in CLBP (Picavet, Vlaeyen and Schouten, 2002; Turk and Okifuji, 2002; Peters, Vlaeyen and Weber, 2005; Quartana, Campbell and Edwards, 2009). The NeuPSIG (Special Interest Group on Neuropathic Pain) guidelines on CLBP and PNP assessment in daily practice and clinical trials recommend measuring kinesiophobia and pain catastrophizing (Haanpää et al., 2011). Psychosocial

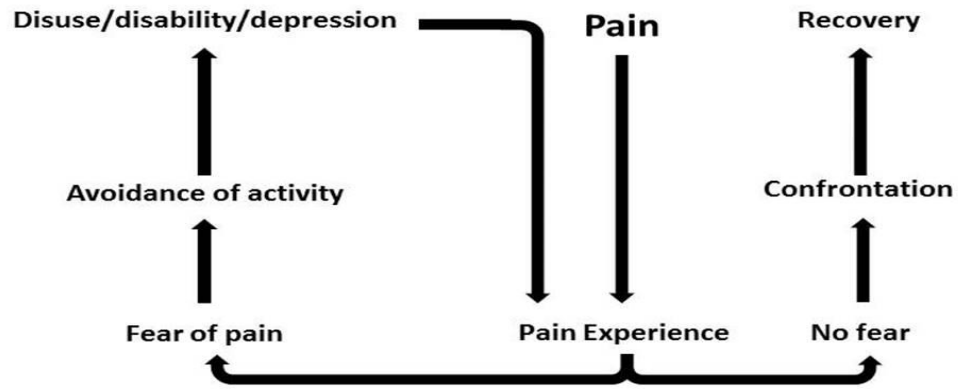
factors are recognized as important moderators of the pain experience and may lead to different treatment responses in various patient phenotypes (Turk and Okifuji, 2002).

Kinesiophobia

Kinesiophobia is defined as an irrational and debilitating fear of physical movement and activity resulting from a feeling of vulnerability to painful injury or re-injury (Kori, Miller and Todd, 1990; Celletti et al., 2013). Patients with kinesiophobia tend to avoid movements that they believe may increase pain (Schwellnus et al., 2011). Activity avoidance can result in impaired strength and flexibility, increased pain and depression, helplessness, and disability (Moore, 2010; Schwellnus et al., 2011). Vlaeyen et al., (1995) identified patients with CLBP whose disability was driven by kinesiophobia and not nociception. This important finding demonstrated that diagnosis and treatment should include examining kinesiophobia associated with CLBP and not only the report of pain.

The scale most used, internationally, to measure kinesiophobia is the Tampa Kinesiophobia Scale (TSK). The scale measures fear of movement/ (re)injury and is often used in studies, supporting the fear avoidance model (FAM) (Figure 5) (Vlaeyen et al., 1995; Nicholas et al., 2012). This model describes patients who acquire a fear of movement and chronic disability after an initial acute phase of pain (Crombez et al., 2012). In a review of related measures to pain-related fear, constructs of fear avoidance, fear of movement and kinesiophobia are identified albeit having unsupported construct validity (Lundberg et al., 2011). Self-reflection should be noted when choosing the TSK as an outcome measure since therapist's own fear-avoidance beliefs may impact on their patient's LBP outcomes (Coudeyre et al., 2006). Authors concur, rationale for treatment of kinesiophobia could target risk factors to facilitate rehabilitation (Vlaeyen et al., 2002; Pincus et al., 2010).

Figure 5: The fear avoidance model (Vlaeyen et al., 1995).



Therapeutically, graded activity interventions have shown a reduction in kinesiophobia (Van Den Hout et al., 2003; Sullivan and Stanish, 2003). A study of 33 patients with CLBP and a systematic

review of 37 articles on pain related fear, 13 of which were specific to kinesiophobia, concur that strategies can be developed to reduce kinesiophobia in CLBP (Vlaeyen et al., 1995; Lundberg, 2011).

Pain catastrophizing

Pain catastrophizing is defined as an exaggerated, negative mental state that can occur in patients in response to an actual or anticipated painful experience (Sullivan et al., 2001a). Pain catastrophizers are known to report higher pain intensities and disability (Sullivan and Stanish, 2003). A reduction in catastrophizing from baseline measures may demonstrate a minimising of pain and disability (Sullivan et al., 2006a; Adams et al., 2007).

Since catastrophizing can exaggerate pain, it is thought to be a coping mechanism related to acute pain. Thus, catastrophizing could be considered helpful unless the patient develops a chronic pain condition (Sullivan, Adams, and Sullivan, 2004). To mitigate pain when applying a given treatment, understanding theoretical models underpinning catastrophizing may assist the therapeutic application. These models include the Beckian model of cognitive errors (Beck, 1995), an appraisal process resulting in heightened attention to pain (Eccleston and Crombez, 1999), and a social coping mechanism (Sullivan, Adams, and Sullivan, 2004). The Beckian model suggesting catastrophizing as a cognitive error proposes treatment using cognitive restructuring, replacing identifiable automatic maladaptive cognitions with realistic and rational thinking (Beck, 1995). If catastrophizing involves an appraisal process causing greater attention to pain, expectation of increased pain and threat perception, then a therapeutic proposal is to modify attentional focus towards distraction and positive coping strategies (Eccleston and Crombez, 1999). Catastrophizing was proposed as a coping mechanism to gain social support (Sullivan, Adams, and Sullivan, 2004). Ideally this may solicit empathy socially but may unfortunately trigger, maintain, or reinforce exaggerated pain expression (Sullivan et al., 2001a). It was suggested that the relation between central nociceptive mechanisms is bi-directional (Sullivan et al., 2001a, 2001b). Interventions to mitigate catastrophizing require incorporating treatments to assist catastrophizers to disengage their attention from painful symptoms (Sullivan et al., 2001a).

Patients who catastrophize about their pain, often have difficulties in using cognitive attention diversions strategies such as distraction using positive coping strategies, to help decrease their sensation of pain (Heyneman et al., 1990; Crombez et al., 1998). Excessive focus on nociception or pain sensations may allow facilitation of pain access into consciousness and pain magnification (Eccleston et al., 1997; Eccleston and Crombez, 1999). Catastrophizers' central neural mechanisms might become more sensitized by engaging in cognitive activities which amplify pain signals (Sullivan, 2001b). Catastrophizers are shown in neuro-imaging studies to have significantly more activated areas responsible for attentional modulation in painful experiences (Gracely et al., 2004;

Seminowicz and Davis, 2006). Concurring evidence shows attentional focus appears to be the common conduit for cognitive and affective variables influencing the chronic pain experience (Gracely et al., 2004; Seminowicz and Davis, 2006; Sullivan et al., 2009).

Clinical research shows pain catastrophizing is a modifiable variable and fundamental in interventions facilitating recovery or chronic pain adaptations (Jensen, Turner and Romano, 2001; Keefe, Abernethy and Campbell, 2005; Sullivan et al., 2005a; Smeets et al., 2006a; Smeets et al., 2006b; Smeets et al., 2006c). Ten weeks (50 hours) treatment involving exercise and psychosocial treatment led to reduced pain catastrophizing in 75 patients with whiplash (Adams et al., 2007). A multidisciplinary pain treatment program used in a RCT with 214 patients with non-specific spinal pain, showed up to 40% reduction in Pain Catastrophizing Scale (PCS) scores in 3 weeks (82 hours) (Jensen, Turner and Romano, 2001). Similarly, decreases in catastrophic thinking range between 24% and 27% in two physical therapy intervention studies done over ten and four weeks respectively (Sullivan et al., 2006c; Sullivan et al., 2008). However, Smeets et al., (2006a) found no significant decrease in catastrophizing between physiotherapy, cognitive and combined treatments. A recent systematic review of 79 studies of musculoskeletal pain showed the best evidence (moderate-high quality) was found for cognitive-behavioural therapy, multimodal treatment, and acceptance and commitment therapy (Schűtze et al., 2017).

Clinically, rehabilitating pain catastrophizers may have limitations too. Concurring evidence shows catastrophizers reported poor analgesic effects with an opiate pharmacological intervention, and topical analgesics in pain catastrophizers, resulting in increased pain responses with PNP sufferers (Haythornthwaite et al., 2003a; Haythornthwaite et al., 2003b; Fillingham et al., 2005; Mankovsky et al., 2012). Suggested mechanisms involved in these negative responses are an endogenous nocebo effect due to negative cognitions (Fillingim et al., 2005). Further negative responses are attributable to compromised descending pain inhibition (Edwards et al., 2006; Weissman-Fogel, Sprecher and Pud, 2008; Goodin et al., 2009).

The PCS is criticized as being underdeveloped (Quartana, Campbell and Edwards, 2009). However, on balance it is a broad assessment of catastrophic thinking in individuals in homogenous pain, as well as in neuropathic pain (Picavet, Vlaeyen and Schouten, 2002; Sullivan et al., 2009; Haanpää et al., 2011). Treatment rationale for catastrophizing proposes implementation of risk factor targeted interventions to facilitate CLBP rehabilitation (Sullivan et al., 2009).

Despite catastrophizing being recognized as a useful modifiable outcome for CLBP, it was not utilized in the reviews using walking to treat CLBP (Hendrick et al., 2010; Lawford, Walters and

Ferrar 2015; Sitthipornvorakul et al 2018). By and large, a systematic review recommended further research in measuring CLBP psychosocial outcomes in relation to walking (Hendrick et al., 2010).

2.6 Treatment

Aside from numerous physiotherapy modalities reviewed by the American Pain Society (Table 8) used worldwide, a variety of treatments are available for CLBP (Chou et al., 2007). These include traditional medical management (mainly via general practitioners and pharmacotherapy), self-management programs, and interventional pain management programs (Airaksinen et al 2006; Chou et al, 2007; Vargas-Schaffer, G., 2010; Krein et al, 2013). In an article reviewing systematic reviews and randomised controlled trials of physical treatments for CLBP, exercise, massage and spinal manipulations were shown to be effective, with exercise having the most durable effect (Maher, 2004). CLBP treatment guidelines from 13 countries were similar and recommend medication, manipulation, supervised exercise therapy, cognitive behavioural therapy (CBT), and multidisciplinary treatment (Koes et al., 2010). These guidelines exclude South Africa which currently has no guidelines for CLBP. A South African physiotherapy study showed reasons for the selection of the treatment modalities were undergraduate education received, individual clinical experience and postgraduate course attendance (Naidoo et al, 2012). Data on CLBP treatment in low-income countries are scarce (Omokhodion and Sanya, 2003).

Table 8: Non-pharmacological therapies for CLBP were reviewed by the American Pain Society and American College of Physicians (Chou et al., 2007).

Non-pharmacological therapies for CLBP	Acupuncture
	Spinal joint manipulation
	Exercise therapy
	Massage
	Interdisciplinary therapy
	Functional restoration
	Inter-ferential therapy
	Low-level laser
	Short wave diathermy
	Superficial heat/ cold therapy
	Lumbar supports
	Transcutaneous electrical nerve stimulation
	Therapeutic ultrasound
	Traction
	Psychological therapies
	Yoga

Between 2010 and 2018 three systematic reviews were conducted comparing an array of combined physiotherapy modalities (manipulation, massage, education, supervised exercise, self-treatment, electrotherapy) to a variety of walking programs (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). A systematic review on CLBP treatment concludes that exercise therapy had the most durable effect on CLBP (Maher 2004). However, two reviews comparing walking exercise therapy to usual care physiotherapy concurred that there was low quality evidence that walking as exercise therapy was as effective as various combinations of physiotherapy modalities in improving pain and disability in CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015). RCT's incorporated effective modalities of walking as exercise, massage, and spinal manipulations (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). No study had yet compared walking on its own to stabilization exercise, massage and manipulation; and to a combination of these. Furthermore, descriptions of usual care physiotherapy in three reviews did not allow for comparison between studies (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). Modalities used in usual care physiotherapy differed between the primary studies in the reviews. Similarly, there was a wide variety of walking exercise interventions used in the three reviews (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). Various forms of therapeutic walking exercise (treadmill, over ground, using stairs) and different protocols of administration (pedometer-based, heart rate based, time based without objective measure, un/supervised) may have resulted in differing conclusions regarding the use of walking as exercise. However, no study using walking as a treatment for CLBP has yet described pain phenotypes in the participants.

Authors concur, with PNP resistant to analgesia, PNP patients exhibit heightened pain intensities, impacting negatively on their physical, psychological, and social functioning (Turk and Okifuji, 2002; Chetty et al., 2012). Most treatment recommendations for neuropathic CLBP are pharmacological, with minimal physiotherapy modality advice (Chetty et al., 2012; Baron et al., 2016). 'Specialized physiotherapy' is recommended as a treatment for neuropathic CLBP (Chetty et al., 2012; Baron et al., 2016). However, albeit numerous modalities are available, there is no definition of 'specialized physiotherapy' (Chou et al., 2007). Conflicting evidence is seen where the UK NICE 2016 guidelines do not recommend transcutaneous electrical nerve stimulation (TENS) for CLBP treatment. However, the modality is recommended in other neuropathic guidelines (Chetty et al., 2012; Baron et al., 2016). This further demonstrates the gap in knowledge CLBP pain phenotypes in physiotherapy.

Unlike physiotherapy modalities, pharmacological guidelines acknowledge that nociceptive and neuropathic CLBP are separate entities (Pergolizzi et al., 2008; Romanò, Romanò and Lacerenza,

2012; Chetty et al., 2012; Baron et al., 2016; National Institute for Health and Care Excellence, 2019). Despite the development of guidelines, pharmacological treatment has limitations for PNP and NP phenotypes, including, inefficiency, inadequacy or adverse effects (Chou, Clark and Helfand, 2003; Kalso et al., 2004; Moore and McQuay, 2005). Including pain phenotypes in modelling of physiotherapy studies has yet to be explored. A cross-sectional study conducted in South-Africa examined the role of spinal manipulation, massage, and isometric lumber stabilisation exercises on patients with CLBP commented on a dearth of knowledge regarding pain phenotypes in South African physiotherapists (Naidoo et al., 2012).

Physiotherapy treatment / usual care

A number of different therapies (Table 8) can be used by physiotherapists for the treatment of CLBP (Chou et al., 2007; van Middelkoop et al., 2011). CLBP responds favourably to spinal manipulation, massage and isometric lumbar stabilization exercise and advice (Maher, 2004; Liddle, Baxter and Gracey, 2009; Clenzos, Naidoo and Parker, 2013; Naidoo et al., 2012).

Physiotherapy modalities chosen by South African physiotherapists included general exercises (30%); spinal manipulation (28%); massage (18%); education (12%) and exercising core stabilizing muscles (12%) (Naidoo et al., 2012). A national survey of 419 Irish physiotherapists demonstrated whilst also using manipulation and massage, that advice and specifically exercising core stabilizing muscles were most frequently used to treat CLBP (Liddle, Baxter and Gracey, 2009). A review which advocated exercise more than manipulation and massage to treat CLBP, expressed that the limitation in exercise was generic prescription (Maher 2004). For example, Liddle, Baxter and Gracey (2009) showed that Irish physiotherapists preferred prescribing core stabilization to flexibility exercise and aerobic exercise (such as walking). Pain intensity but not pain phenotype was considered when prescribing these types of exercise. Furthermore, 207 specialist physiotherapists in South Africa completed questionnaires acknowledging that assessment of pain prior to treatment was inadequate (Clenzos, Naidoo and Parker, 2013). This possibly highlights that CLBP management may require exploration of pain phenotypes.

A study of 489 CLBP patients living in South Africa was completed and concluded that CLBP management in South Africa appears ineffective due to 90% of patients receiving medication only, minimal physiotherapy referral and no guideline base to treat CLBP (Major-Helsloot et al., 2014). NHS NICE 2016 guidelines and 2007 Austrian guidelines recommend the use of mobilization, massage and exercise for CLBP (Liddle, Baxter and Gracey, 2009; Koes et al., 2010; National Institute for Health and Care Excellence, 2016). Despite widespread use of these modalities in CLBP, sub-grouping through patient screening and pain phenotyping is not explicit in the review of national guidelines in Koes et al., (2010). Neurological screening was based largely on a straight leg raise test

in Koes et al., (2010) and use of the STarT Back risk assessment tool (National Institute for Health and Care Excellence, 2016). This may not be sufficient in phenotyping CLBP.

Due to the paucity of evidence with physiotherapists using pain phenotyping, exploration of CLBP phenotyping may provide insight into these patients' presentations.

Spinal manipulation

Spinal mobilization comprises of low-velocity passive movements within or at the limit of joint range (Koes et al., 1996; Ruddock et al., 2016). Spinal manipulation though is defined as a high velocity thrust to a joint past its restricted range of movement (Koes et al., 1996; Ruddock et al., 2016). Harvey et al., (2003) shows most systematic reviews do not distinguish between manipulation and mobilization, as is seen in Lawford, Walters and Ferrar (2015) and Hendrick et al., (2010). This is commonly referred to as spinal manipulation/ manual therapy. Manipulation of neural structures may have analgesic effects in neuropathic pain (Vigotsky and Bruhns, 2015). However, care should be taken when using manipulation for neuropathic pain as the mechanical stimulus may increase sensitization (Gosling, 2013; Courtney, Fernández-de-Las-Peñas and Bond, 2017).

Manipulation improves spinal mobility and potentially has immediate effects of relieving LBP (Chiradenant et al., 2003; Goosens et al., 2005; Bokarius and Bokarius, 2010; Ruddock et al., 2016). A review of any joint manipulation in the body concurred with a narrative review which included six studies on spinal manipulation (Savva et al., 2014; Vigotsky and Bruhns, 2015). Joint manipulation has an important role in post treatment hypoalgesia. Both reviews argue the analgesic mechanism involved increases pain tolerance and decreases sensitization which is neurophysiological in nature through descending modulation circuits (Savva et al., 2014; Vigotsky and Bruhns, 2015). Clinically, different operator velocities and spinal-level locations during manipulation elicit varied descending pain modulation mechanisms (Vigotsky and Bruhns, 2015). The varied methodologies, use of rat models and human models, and small sample sizes highlighted in this review leads to the conclusion that exact analgesic circuitry in human populations remains unknown (Vigotsky and Bruhns, 2015). The authors acknowledge this uncertainty regarding the circuitry involved and the confounding effect of touch (when using manipulation) and placebo, which are known to share endogenous opioid and endocannabinoid analgesic mechanisms (Vigotsky and Bruhns, 2015). The Pain Gate theory (Melzack and Wall 1965) is still applied currently where another review of physiotherapy treatment maintains skin touch stimulates Aβ mechanoreceptors following a proprioceptive stimulus. This assists with inhibition of painful stimuli in the CNS (Gosling, 2013). Manipulation results in opioid activation and endorphin release, as well as non-opioid (serotonin, norepinephrine, dopamine, GABA, and growth hormone) neurotransmitter and hormone release (Gosling, 2013). In this review

of 38 articles from 1997 to 2011, sources of data and methods of use in contributing studies were not identified, but summaries of the mechanisms by which physiotherapy techniques reduced pain were.

Two reviews concurred manipulation did not significantly reduce pain (Avery and O'Driscoll, 2004; Rubinstein et al., 2011). The review not supporting manipulation included three RCTs but did not mention how the studies were selected (Avery and O'Driscoll, 2004). Another review included 26 RCTs, with a comprehensive search methodology (Rubinstein., et al; 2011). Although Rubinstein., et al (2011) found high quality evidence that manipulation has a small and significant effect on short term CLBP pain relief, the authors concluded that this was not clinically relevant due to inefficiency of short term relief on the chronic nature of CLBP. However, the UK evidence report supported manipulation for effectively treating CLBP (Bronfort et al., 2010). The UK evidence report supported the use of manipulation through five systematic reviews, including 70 RCTs (Bronfort et al., 2010). The reviews found manipulation superior to sham interventions. Since auditory, touch and movement cues are similar between real and sham manipulation, there is little agreement among experts as to what constitutes an effective sham manipulation. However, there is some evidence as to what may be acceptable as an effective sham manipulation of the lumbar spine. Described in Hancock et al., (2006), "the trunk and pelvis are then rolled together so no lumbar inter-vertebral motion occurs". Additionally, three internationally used guidelines recommend the use of manipulation. These include: NICE, the American College of Physicians, American Pain Society, and the European guidelines for CLBP (National Collaborating Centre for Primary Care [UK], 2009; Chou et al., 2007; Airaksinen et al., 2006).

Massage

Massage is defined as soft tissue manipulation using one's hands or a mechanical device (Furlan et al., 2002). Massage is applied as a sole treatment or in combination with other treatments, with techniques varying between effleurage, petrissage, friction, hacking and kneading (Maher, 2004; Airaksinen et al., 2006).

Several mechanisms of action implied in massage include decreasing muscle tone and fatigue, diffuse noxious inhibitory controls (DNIC), and pain gate theory (Ernst and Fialka, 1994; Vigotsky and Bruhns, 2015; Melzack and Wall 1965; Gosling, 2013; Field, 2014). Reviews on physiotherapy techniques concur gentle massages may activate Aβ fibres inhibiting nociceptive input via Aδ and C fibres (Gosling, 2013; Vigotsky and Bruhns, 2015). Massage may activate non-opioid substances namely serotonin, norepinephrine, dopamine, GABA, and growth hormone which inhibit nociception (Gosling, 2013). The reviews which supported the use of massage failed to define explicit methods used to determine how articles were included (Gosling, 2013; Vigotsky and Bruhns, 2015). A five-week comparison study of skin contact versus Swedish massage (n=45) and four papers reporting on

pleasure and analgesia related to human touch and interaction, concur on the therapeutic nature of human touch (Rapaport, Schettler and Bresee, 2012; Ellingsen, 2014). Skin contact elevates oxytocin levels, functioning as a somatosensory system analgesic (Rapaport, Schettler and Bresee, 2012; Ellingsen, 2014). Albeit not studying CLBP patients, participants in both studies were treated with non-noxious touch. Clinically relevant, a dose dependant relationship was highlighted with bi-weekly massage being more potent than once a week (Rapaport, Schettler and Bresee, 2012). Findings in four papers acknowledge dose dependant relationships again, between increased frequency of gentle massage and increasing pleasure or pain reduction, enhancing the positive effects of oxytocin (Ellingsen, 2014). However, no large effect-size differences for neuroendocrine measures on pain were observed between once-weekly and twice-weekly massage sessions (Rapaport, Schettler and Bresee, 2012). The frequency of massage and effect size in pain reduction for CLBP may warrant further investigation.

Despite evidence supporting massage as a CLBP treatment, a systematic review of systematic reviews concludes massage has limitations (Kumar, Beaton and Hughes, 2013). Massage was not recommended in the 2004 European guidelines for NSCLBP (Airaksinen et al., 2006). However, massage was reported as an effective therapy for CLBP in the UK evidence report and is included in the 2016 UK NICE guidelines treatment package for LBP (Bronfort et al., 2010; National Institute for Health and Care Excellence, 2016). Depending on the context of the organizational infrastructure which supports or refutes the use of massage, it is arguable as to its appropriateness as a treatment choice.

Two systematic reviews of high quality (Ernst, 1999; Furlan et al., 2002) were used in the 2004 European guidelines for NSCLBP (Airaksinen et al., 2006). Eight RCTs formed the basis of the evidence in one review Furlan (2002). Massage therapy was compared to a variety of treatments and limited evidence showed massage was more effective in improving pain outcomes than sham procedures (Preyde, 2000), remedial exercise and posture education (Preyde, 2000), acupuncture (Cherkin et al., 2003), and general physical therapies (Hsieh et al., 1992, Pope et al., 1994). A systematic review including 25 RCTs specifically assessed the effects of massage for CLBP and acute LBP (Farber and Wieland, 2016). The review compared massage to sham therapy, waiting list/ no treatment, manipulation, mobilization, TENS, acupuncture, traction, relaxation, physical therapy, and exercises or self-care education (Farber and Wieland, 2016). The authors concluded reduced disability benefits for CLBP patients using massage when compared to sham therapy or waiting list.

A systematic review concurred with an updated Cochrane review, that benefits of massage include decreased pain and improved functional status, especially if combined with exercise and CLBP education (Ernst, 1999; Furlan et al., 2008). The review of 13 randomised and quasi-randomised

trials associated benefits depending on massage technique (Furlan et al., 2008). Acupuncture massage, defined as tonic stimulation of entire meridians, was more beneficial than classical, Swedish or Thai massage (Furlan et al., 2008). Since only two studies in the review compared different techniques of massage, this may warrant further exploration. Despite examining the evidence for massage as a treatment for a variety of musculoskeletal (CLBP, LBP, cervicogenic headache) and non-musculoskeletal conditions (fibromyalgia, asthma), conclusions were made that massage is an effective treatment for CLBP (Bronfort et al., 2010). The report included 49 recent relevant systematic reviews, 46 RCTs not included in other systematic reviews and 16 evidence-based clinical guidelines. Due to the robust nature of the systematic review described in the previous paragraph (Farber and Wieland, 2016), evidence report (Bronfort et al., 2010), and Cochrane review (Furlan et al., 2008), massage is likely to be clinically supported.

Core stabilization intervention exercise program

The most common form of exercise treatment for CLBP by SA and UK physiotherapists is to use spinal stabilization exercises (Liddle, Baxter and Gracey, 2009; Naidoo et al., 2012). The core or spinal pelvic complex is a multi-segmental structure consisting of a thoracic cage, pelvic rim, five lumbar vertebrae, and hip joints and associated soft tissues. Typical of the motor control model of CLBP classification, the wide-spread use of stabilization exercises is based largely on a hypothesis that there is a link between activation and timing of local spinal stabilizing muscles and retardation of LBP (Richardson, Hodges, and Hides, 2004). Authors concur that working synergistically, lower back muscles, abdominal muscles, connective tissue between trunk muscles and lumbar spine neuromuscular control are thought to generate and control motion (Richardson, Hodges and Hides, 2004; Behm et al., 2010). A meta-analysis of 16 CLBP studies published before 2011 showed motor control exercise or stabilization given for an average of eight weeks was superior to other forms of exercise with intermediate term pain relief and improved functional outcomes (Byström, Rasmussen-Barr and Grooten, 2013).

A review demonstrates the success of core strengthening on CLBP pain and disability reduction (deducing core stabilization and conventional exercise independently both lower CLBP) (Gordon and Bloxham, 2016). The review based on biomechanical theory, identified distinct muscles involved in increasing spinal stability (Table 9).

Table 9: Core lumbar spine abdominal bracing muscles (Gordon and Bloxham, 2016).

Core lumbar spine abdominal bracing muscles	
Abdominal muscles	**Lower back muscles**
Rectus abdominus	Psoas Major
Obliquus externus abdominus	Interspinales and Intertransverarii
Obliquus internus abdominus	Quadratus Lumborum
Transversus abdominus	Lumbar multifidus
	Longissimus thoracis pars lumborum
	Longissimus thoracis pars thoracis
	Iliocostalis lumborum pars lumborum
	Iliocostalis lumborum pars lumborum

This biomedical approach supports the hypothesis that muscular hypertrophy and spinal stiffness are beneficial to the spine (Table 10) (Gordon and Bloxham, 2016).

Table 10: Muscular strength and stabilization programs for NSCLBP patients (Gordon and Bloxham, 2016).

Core exercise used	Length of intervention	Study type	Outcome measures	Findings of the study	Population group	Author
Using an unstable standing surface and performing unexpected upper limb movements.	8-week	Pilot study	Pain Visual Analogue Scale	39.5% significant reduction in pain post intervention	10 NSCLBP patients (3 male, 7 female)	(Ŝarabon, 2011)
Core exercises using slow curl ups, bird dog, plank, sit ups compared with conventional exercise using stretching of tight muscles	12-week	RCT	Pain Visual Analogue Scale	Post intervention, reductions in Pain Visual Analogue Scale were 76.8% in the Experimental group and 62.8% in the Control group	30 NSCLBP patients (20 male, 10 female)	(Inani and Selkar, 2013)
Strengthening of transversus abdominus and internal oblique muscles alone and compared to strengthening together with ankle dorsiflexion against resistance of a rubber band (experimental group) to enhance core strengthening. 10 sets of 20 seconds.	8-week	RCT	Pain Visual Analogue Scale Pain Disability Index Visual Analogue Scale Pain Rating Scale	Post intervention significant reductions in Experimental groups Pain Visual Analogue Scale of 32.5%, 23.2% Pain Disability Index, and 21.5% in pain rating scale. The Control group post intervention had significant reductions in Pain Visual Analogue Scale of 16.8%, 12.4% in Pain Disability Index and 8% in Pain Rating Scale.	40 NSCLBP patients (19 male, 21 female)	(You et al., 2014)
Core muscular strength programme was compared to core stability programme	4-week	Randomised clinical open-label study	Oswestry Disability Index Short-Form 36	Post intervention, significant reduction of 53% in the experimental group and 14% reduction in the control group.	160 NSCLBP patients (63 male, 97 female)	(Stankovic et al., 2012)
Exercises to improve lumbar stability. Abdominal curl ups with a slight rotation and a squat. This program involved education and correction of lifting techniques	12-month	RCT	Visual Analogue Scale	Significant reduction of 39% in pain	106 middle aged working men reporting an episode of NSLBP within the previous 3 months without severe disability.	(Suni et al., 2006)
Muscular strength program used with varied inversion traction angles.	8-week	Clinical trial	Visual Analogue Scale	Significant reductions in Pain Visual Analogue Scales of 61.6% in both inversion groups of -30° and -60°. Significant reduction of 34.9% in the supine group.	47 women with NSCLBP	Kim et al., 2013.

The biomedical approach to using abdominal muscles to support the lumbar spine in potentially treating CLBP is inconclusive into how it can specifically be applied as exact lumbar stabilization exercise protocol is seldom described. Furthermore, mechanical models may be limited in clinical application. Three studies including asymptomatic participants concur that abdominal bracing and strengthening have generic spinal stiffening effects (Stanton and Kawchuk, 2008; Stokes, Gardner-Morse & Henry, 2011; Katsura et al., 2011). If muscular hypertrophy and spinal stiffness are clinically meaningful, then these studies support this as treatment. In 28 asymptomatic participants, strengthening was said to significantly increase posterior-anterior spinal tissue stiffness ($p<0.05$) thereby improving core stability through dampening lumbar stresses (Stanton and Kawchuk, 2008). Using a biomechanical model of the spine and its musculature, spinal stability is not significantly influenced by selective abdominal muscle activation (Stokes, Gardner-Morse & Henry, 2011). Forcing either transversus, obliques, or rectus to be preferentially active did not systematically increase stability, however generic intra-abdominal pressure increased stability (Stokes, Gardner-Morse & Henry, 2011). No patterns associating forced activations with either an increase or a decrease in stability were identified. Hydrotherapy programs in women exercising the core compared resistance versus no resistance, and demonstrated significant improvements in abdominal strength and function, together with abdominal muscular hypertrophy, in the resistance group (Katsura et al., 2011). However, the studies using human participants described above exclude psychosocial measures, which if measured may have highlighted the psychosocial effects of lumbar stabilization. which may provide more information of the mechanism by which the stabilization exercises worked.

Following this, varied exercises appear to have generic mechanical benefit for symptomatic persons with CLBP. A systematic review and meta-analysis which included 22 studies of patients with LBP and CLBP examining disability and pain outcomes compared stabilization exercise to other forms of exercise such as stationary cycling and using slings (Smith, Littlewood and May, 2014). Stabilization exercises were the sole intervention for the majority of studies, five studies examined patients treated individually, nine studies involved exercises done as group sessions, and three studies combined with general exercises or electrotherapy (Smith, Littlewood and May, 2014). The review concluded that stabilization is no more effective than any other form of active exercise in the long term. Furthermore, when using psychosocial outcomes, no benefits were seen in terms of fear avoidance when stabilisation exercises were compared to stationary bike exercise, sling, or general exercises (Unsgaard-Tøndel et al., 2010). There has been some suggestion that stabilization exercise promotes unhealthy beliefs and kinesiophobia (Nijs et al., 2013).

Despite exercise recommendations for the treatment of CLBP, the specific ideal exercise type remains inconclusive (Koes et al., 2001; American College of Sports Medicine, 2013). Studies have often used different methodology for stabilization exercises (Table 10). Several studies on CLBP

pain outcomes, show variability in core stabilization exercise methodology (Chon, Chang and You, 2010; Katsura et al., 2011; You et al., 2014). This could impact on the effectiveness of this type of exercise in patients with CLBP and associated psychosocial outcomes.

However, Table 11 demonstrates that several studies estimate core stabilization success is dependent on patient selection based and objective mechanical tests, and not on stabilization exercise methodology (Hicks et al., 2005; Gazzi et al., 2014). The concentration on patient selection also appears to be based on mechanical classification.

Table 11: Four variables influence CLBP stabilization exercise program success (Hicks et al., 2005).

If three or more are present, then it predicts stabilization exercises being successful in decreasing CLBP.
Age (under the age of 40 increases the treatment success)
An Active straight leg raise test higher than 91 degrees
Presence of aberrant movement during lumbar range of movement
Presence of a positive prone instability test

Despite the use of stabilization exercise as an exercise-based treatment for CLBP in the UK and SA, there seems insufficient evidence in reviews to recommend its use in preference to other exercise forms (Smith, Littlewood and May, 2014; Wang et al., 2012). Examination of biopsychosocial outcomes is highlighted when exploring exercise to treat CLBP. Other forms of exercise such as walking merit exploration for the treatment of CLBP.

Walking used as physical activity, and exercise

Recognizing that walking has application in health research as discriminant PA and exercise is salient. Physical activity (PA) is a broad term used to describe any bodily movements that results in energy expenditure (Caspersen, Powell and Christenson, 1985). Exercise is defined as structured and planned physical activity (Caspersen, Powell and Christenson, 1985; Winter and Fowler, 2009; Tremblay et al., 2017). However, often the terms exercise and PA are used interchangeably. They are discretely different (Table 12). Furthermore, people can be both physically active and sedentary (Thivel et al., 2018). According to this recent classification, a previous study using a walking intervention categorised inactive participants as sedentary (Krein et al., 2013).

Table 12: Definitions of Physical activity and Sedentary behaviours (Thivel et al., 2018)

Terms	Definitions
Physical activity	Body movements generated by the contraction of skeletal muscle that raises energy expenditure above resting metabolic rate. It is characterized by its modality, frequency, duration, and context of practice.
Physical inactivity	Represents the non-achievement of physical activity guidelines
Exercise	Subcategory of physical activity that is planned, structured, and repetitive, and favours physical fitness maintenance or development.
Sport	Sport is part of the physical activity spectrum and corresponds to any institutionalized and organized practice, reined over specific rules.
Sedentary behaviours	Any waking behaviours characterized by an energy expenditure $\leq$ 1.5 METs, while in sitting, reclining, or lying posture.

Walking can be defined as either. It can be purposeful and structured or encompass activities of daily living, such as housework, and yard work. Walking as an activity of daily living PA or exercise has a myriad of general health benefits (Table 13). It has low risk of injury, is considered affordable, requires no training, supervision, or specialized equipment, and has high accessibility (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

Table 13: Health benefits of walking.

Medical condition	Health benefit of walking	References
Chronic Lower Back Pain	Walking according to American College of Medicine guidelines is shown to improve pain, disability and fear of movement outcome measures	Eadie et al., 2013; Hurley et al 2015; American College of Sports Medicine, 2011
Dementia	Deceased depression and decline in dementia associated with walking.	Kvæl, Bergland and Telenius, 2017
Diabetes	Walking ≥7,500 steps/day, of which ≥3,000 steps (representing at least 30 min) should be taken at a cadence ≥100 steps/min can reduce the risk of type II diabetes.	Tudor-Locke and Schuna, 2012
Heart disease	Prevalence of any walking decreased with increasing CVD risk Cardiovascular disease risk decreased with any walking done including leisure time walking, 12-week walking programs may assist with decreasing total cholesterol mitigating heart disease.	Omura et al., 2019; Coghill and Cooper, 2008; American College of Sports Medicine, 2011; Rodriguez-Sanchez et al., 2014
Arthritis	Walking can reduce pain and improve function, mobility, mood, quality of life, without worsening symptoms	Bruno et al., 2006; Katz et al., 2018
Depression and anxiety	Walking facilitates endorphin release, promoting relaxation, preventing anxiety and depression	Bruno et al., 2006
Alzheimer's disease	Walking facilitated exercise adaptations improving sleep and quality of life in Alzheimer's patients	McCurry et al., 2011
Weight loss	Walking 20 minutes per day will metabolize 7 pounds of body fat per annum. Women walking 1 hour per day, 5 times per week with a regulated diet can lose 25 pounds and maintain the weight loss	Richardson et al., 2008, American College of Sports Medicine, 2011
Prostate cancer	Lower mortality risk, decreased side effects, improved function quality of life and weight loss.	Champ et al., 2016; Gerriten and Vincent, 2016
Colon cancer	Walking regularly following the ACSM guidelines reduces cancer related fatigue, improves quality of life and function in colorectal cancer patients.	Oruç and Kaplan, 2019
Longevity	Walking for more than one hour per day extended life expectancy.	Nagai et al., 2011
Immune function	Short acting boost in immune function	Nieman et al., 2005; Kimura et al., 2006
*1 mile= 1.6 kilometres		

Concurring authors show that walking has been categorized as an aerobic exercise by a survey by Irish physiotherapists on the exercise advised and provided to their patients for CLBP, a systematic review of aerobic exercise for reducing CLBP, and a review on walking to treat CLBP and (Liddle, Baxter and Gracey., 2009; Privett, 2012; Lawford, Walters and Ferrar., 2015). A review on walking as a treatment for CLBP, also defines exercise as structured and deliberate PA to improve health and specifically CLBP (Lawford, Walters and Ferrar, 2015). This highlights the context of application of walking. It can be advised for mobility and activities of daily living however physiotherapists can deliver walking interventions that are planned and structured repetitively to achieve a change in outcomes.

As a consequence of the health benefits from PA, evidence predicts a reduction of at least 50% mortality in highly fit people than people with low fitness (Warburton, Nicol and Bredin, 2006). However, physical inactivity is associated with the development of several different long-terms conditions and diseases, such as those described in Table 14 (Warburton et al., 2007; Pedersen and Saltin, 2015). Insufficient walking in populations can be detrimental to health (Tudor-Locke, Hart and Washington 2009; Hirvensalo et al., 2011; Pillay et al., 2015; Parker et al., 2017; Althoff et al., 2017). With exercise being a subset of PA, converging evidence support both as prevention and treatment for conditions described in Table 14 (Warburton et al., 2007; Moore et al., 2012; Warburton and Bredin, 2017). Authors concur, the dose-response relationship for PA is curvilinear with relatively small increases in levels of PA, resulting in increased health benefits (Warburton et al., 2007; Moore et al., 2012; Warburton and Bendin, 2017).

Table 14: Chronic diseases affected by physical activity.

Chronic diseases mitigated by elements of physical activity and exercise			
Chronic Disease	**Mitigating Effect**	**Studies involving PA**	**Studies involving exercise**
Physical disability	Improving aerobic metabolic status, increased strength and joint range of movement, improved mood	Moore et al., 2012	Parker et al., 2017
Arthritis	Improving joint functioning and neurophysiological pain control	Booth, Roberts and Laye, 2012	Goh et al., 2019
Obesity	Improving aerobic metabolic status	Kumanyika et al. 2008; Weinstein and Sesso, 2006	Lee et al., 2010; Slenz et al., 2004
Depression & anxiety	Improved mood through neurophysiological and psychological effects	Physical activity guidelines committee 2008	Rothon et al., 2010; Hillman, Erickson and Kramer, 2008
Cognitive function	Organise thoughts, manage time, improved decision making	Etnier et al., 2006; Bidzan-Bluma and Lipowska, 2018	Kramer et al., 1999; Colcombe and Kramer, 2003; Colcombe et al., 2006
Coronary artery disease	Increased energy expenditure lowering artery plaques in heart disease	Warburton, Nicol and Bredin 2006; Mora et al., 2007	Deveza, Elkins and Saragiotto, 2017
Diabetes	Improving glycaemic control	Crandall et al., 2008; Warbuton et al., 2006	Kump and Booth, 2005
Osteoporosis	Increasing new bone production with weight bearing	Warburton, Nicol and Bredin, 2006	Engelke et al., 2006
Certain cancers (e.g.: breast, colon)	Lowering inflammation and improved immune function	Monninkhof et al., 2007; Colditz, Cannuscio and Frazier, 1997; Rezende et al., 2018	Gerritsen and Vincent, 2016
Chronic obstructive Pulmonary Disorders	Increasing breathing rates improving cardiorespiratory health	Warburton, Nicol and Bredin 2006; Booth, Roberts and Laye 2012	Kujala et al., 1996; Martinez et al., 2014
Chronic lower back pain	Reduces pain, disability, kinesiophobia and improves function through improved muscular strength, increased cardiorespiratory health and improves psychological wellbeing.	Maher, 2004; Slade, Molloy and Keating, 2009; Lin et al., 2011	Hayden et al., 2005a; Mayer, Mooney and Dagenais, 2008; Slade, Molloy and Keating, 2009; Freburger et al., 2009; Privett, 2012; Kruger and Billson, 2012; Smith, Littlewood and May, 2014; Gordon and Bloxham, 2016

If walking is observed as a PA, it may be necessary to have objective evaluations of peoples PA suffering with CLBP. Increasing PA to reduce pain related disability outcomes from CLBP remains controversial. Systematic reviews aiming to examine the association with PA and CLBP outcomes of pain related disability have opposing conclusions (Lin et al., 2011; Hendrick et al., 2011). A systematic review of 18 studies investigated the role of PA on disability from CLBP and LBP, 14 of which included only participants with CLBP (Lin et al., 2011). Pooled results indicated that those who reported lower levels of activity were most likely to have higher levels of disability (Lin et al.,

2011). Opposing this evidence, a systematic review of 12-studies, with only two studies including participants with CLBP, investigated whether increasing levels of PA above baseline decreased disability (Hendrick et al., 2011). In conclusion, increased PA was not associated with reduced levels of disability in participants with CLBP (Hendrick et al., 2011). Only one cross sectional study (n=13) in the latter review objectively measured PA using accelerometers (Verbunt et al 2001). The former review included five studies using objective measures of PA detailing more evidence supporting associations of low PA and disability in people with CLBP (Verbunt et al., 2001; Bousema et al., 2007; Ryan et al., 2009; van Weering et al., 2009; Huijnen et al., 2009). As seen in the two reviews, objectively measuring PA and increased PA may both account for reporting reduced self perceived disability. It has been observed when pedometers are used, and improved health outcomes are noted (Krein et al., 2013; Harris et al., 2015).

Measuring devices should be valid for specific PA outcomes. Measuring steps is valid if steps are being compared within or between treatments. Equal comparison of PA between studies may not be fully observed due to interventions including varied methodologies of structured repetitive exercise in both reviews (Hendrick et al., 2011; Lin et al., 2011). Exercises included in the reviews on PA included lumbar stabilization or strength programs (Hendrick et al., 2011; Lin et al., 2011). Step measurements may not be ideal measurements of PA for these interventions. Exercises may have heterogenous mechanisms related to outcomes of improvement. For example, steps taken are not identical to strength outcomes. Furthermore, studies making use of accelerometers over seven to 14 days may not represent participant behaviour over longer periods of time. If pedometer or accelerometer measurements are to be observed when examining the effect of PA on CLBP, PA type using walking as a treatment may be worth further exploration.

Prior or additional PA may affect CLBP participant outcomes. As made clear in a review of examining how many steps are enough for health benefits, dissociating additional PA done as exercise from daily overall PA should be considered in its effect on health-related outcomes (Tudor-Locke and Bassett, 2004). Objectively measuring PA may be fundamental in treating CLBP. Both reviews did not specify including either insufficiently active or active participants only (Lin et al., 2011; Hendrick et al., 2011). Cohort and cross-sectional studies in the two reviews with participants having high or low levels of PA may display different responses to additional PA. Objectively monitoring of PA at both baseline and done when exercising outside of overall daily PA, may further demonstrate if additional PA executed as walking exercise is affecting outcomes. For example, when using walking to treat CLBP, objective monitoring of steps throughout a study and steps done during a walking exercise intervention, may provide objective evidence for effects on outcome measures. Studies in both reviews when not employing objective measures used recall questionnaires (Lin et

al., 2011; Hendrick et al., 2011). Recall measures used by researchers are subjective to participant recall bias as to changing levels of PA over time.

Exercise as a subset of PA has therapeutic benefits. It is unknown if an optimal objective amount of PA exists for populations suffering with CLBP. This can be examined if structured, repetitive, and objectively monitored PA is delivered as exercise for people with CLBP.

Walking shares challenges with other exercise types. Varied exercise types aside from walking can be used in treating CLBP (Van Tulder et al., 2000; Pedersen and Saltin, 2015). Multiple factors associated with exercise may affect associated outcomes. Exercise duration, frequency and intensity are often used to measure progress in exercise programmes. The mode of supervision can vary from unsupervised, to varied levels of supervision, either in person, on the telephone or via the internet. Further research is required into what mechanisms, patient characteristics, and outcomes when considering exercise types and amounts to beneficially treat CLBP.

Despite the ease to which walking can be delivered as an exercise to treat CLBP, there remain a wide array of exercises supported as a treatment for CLBP as seen in Table 15 (Hayden 2005b; Mayer, Mooney and Dagenais, 2008; Henchoz and Kai-Lik So, 2008; Liddle, Baxter and Gracey, 2009; Naidoo et al., 2012; Smith, Littlewood and May, 2014; Pederson and Saltin, 2015). The role of exercise in treating CLBP has been incorporated into clinical guidelines for example the 2004 European guidelines for NSCLBP and the 2016 UK NHS NICE guidelines (Airaksinen et al., 2006; National Institute for Health and Care Excellence, 2016). The reviews on exercise for CLBP conclude that supervised exercise compared with unsupervised exercise is more effective in reducing CLBP associated pain and disability (Mayer, Mooney and Dagenais, 2008; Henchoz and Kai-Lik So, 2008). Reviews on exercise for CLBP detail numerous factors remain inconclusive in application of exercise (Mayer, Mooney and Dagenais, 2008; Henchoz and Kai-Lik So, 2008). A limitation of systematic reviews and guidelines relating to the use of exercise in treating CLPB includes the lack of homogeneity between the studies. This makes it difficult to determine a definitive delivery for the type, duration, frequency and intensity of exercise required. Table 15 shows multiple subcategories are involved in CLBP exercise management, according to Mayer, Mooney and Dagenais, (2008).

Table 15: Exercise categories used to treat CLBP (Mayer, Mooney and Dagenais, 2008)

CLBP exercise subcategories
Activity as usual (recommendations against physical restrictions)
Aerobic (walking/cycling)
Hydrotherapy
Directional preference (McKenzie)
Proprioceptive/ co-ordination (wobble board, stability ball)
Flexibility (yoga, stretching)
Stabilization (low load exercises specific to spinal and abdominal musculature)
Strengthening (resistance exercises)

Furthermore, exercises are often combined with more than one exercise type. Strengthening/ flexibility, aerobic/ strengthening, aerobic (included postural advice), multimodal (included behavioural, positive coping, ergonomic advice), hydrotherapy, McKenzie exercises, and callisthenics are described as specific exercise to treat CLBP (Liddle, Baxter and Gracey 2004). However, two exercise subcategories are often done together (e.g., strengthening/ flexibility) and the exercise advised for the majority of the time is recognized as the specific exercise (Liddle, Baxter and Gracey 2004). This may contribute uncertainty to whish exercise used was responsible for a change in outcomes. A systematic review of 45 randomised trials showed strength/ resistance and stabilization/ co-ordination interventions demonstrated improved pain outcomes in CLBP patients compared to cardiorespiratory and combined exercise programs (Searle et al., 2015). However, the 2016 NICE guidelines are not explicit in recommending specific exercise to treat CLBP, only that patient subjective preferences should be considered including one's needs, capabilities, and preferences (National Institute for Health and Care Excellence, 2016). These guidelines concur with both a review of 14 studies of exercise intervention programmes for CLBP summarized that aerobic fitness, muscular strength and flexibility are all beneficial, and with Exercise is Medicine advice (Gordon and Bloxham, 2016; Exercise is Medicine, 2019). The systematic review of the effects of exercise on NSCLBP highlights the complexity of advocating specifics since CLBP is recognized as multifactorial and not a homogenous condition (Gordon and Bloxham, 2016). Due to the ease of application of walking as an exercise due to its nature as typical PA, further investigation into its effects may be warranted.

Numerous benefits are ascribed to varied exercise types for people with CLBP. Mechanisms related to aerobic function, strength or motor control, and psychological adaptations, may be responsible for outcome measure changes (Gosling, 2013; Gordon and Bloxham 2016; Sluka et al., 2018). Walking has elements of all these factors (Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018; Yoshiko et al., 2018). Concurring reviews propose aerobic exercise modulates CLBP biomedically in the peripheral tissues by increasing blood flow and nutrients to lower back soft tissues, improving

the healing process, and decreasing stiffness (Gosling, 2013; Gordon and Bloxham 2016). Recognizing the psychological benefits of aerobic exercise, pain perception is decreased if one takes part in 30-40 minutes of aerobic exercise (Mayo and Weissman, 2011; Gordon and Bloxham 2016). Two reviews concurred stating exercises based on muscle strength have benefits on strength, and psychologically decrease kinesiophobia in people with CLBP by increasing the patient's locus of control (Mayer, Mooney and Dagenais, 2008; Dreisinger, 2014). Flexibility, endurance, and balance gains were also seen (Dreisinger, 2014). In summary, a review of physiotherapy treatments explains that the modulation of pain via the somatosensory system is salient (Gosling 2013). It occurs through descending inhibition via opioid activation from aerobic/ regular exercise together with effects on the cerebral cortex in cortical reorganization, psychophysiological, behavioural and placebo effects (Gosling 2013). Since evidence highlights biomedical and psychological mechanisms response to exercise for CLBP, it is not clear if one mechanism in isolation is responsible for CLBP relief. Several mechanisms may be involved with walking used to treat CLBP. Authors agree, mechanisms and therapeutic techniques may range from distraction, mastery over the problem, therapeutic alliance, patient education, and expectations of therapeutic success or failure (Tavel, 2014; Stilwell et al., 2017). The effects of both PA and exercise on CLBP modulation are seen in the central nervous system and in the body's tissues.

Due to the mechanisms involved in exercise having possible effects on aerobic function, strength, and psychological adaptation, consideration of appropriate outcomes is necessary using walking as an exercise. In exercise studies historical focus was on biomedical outcomes including pain, general health, and function. An early review of 43 trials using exercise to treat CLBP, pain, function, work absenteeism rates and overall health improvement outcomes were used (Hayden et al., 2005a). A review of 17 RCT's that used exercise as treatment for CLBP, outcomes used were back specific function, generic health status, pain, work disability and treatment satisfaction (Liddle, Baxter and Gracey, 2004). The results showed an improvement in patient's muscular strength outcomes (Liddle, Baxter and Gracey, 2004). Both reviews show a paucity of psychosocial outcomes such as behavioural support and positive coping strategies (Liddle, Baxter and Gracey, 2004). Recommendations from the reviews were for more high-quality trials using fewer yet essential outcome measures. Latter reviews on the effects of exercise on CLBP concurred that further research is required on the psychosocial effects (Mayer, Mooney and Dagenais, 2008; Henchoz and Kai-Lik So, 2008). A review of exercise induced analgesia incorporates improving psychosocial outcomes namely kinesiophobia and catastrophic thinking, although the review suggests the combination of exercise and other treatments may be more efficient in reducing pain (Sluka et al., 2018).

The dosage of physiotherapy and associated CLBP exercise currently remains a combination of evidence, experience, and common sense. There is also limited evidence of how treatment frequency

correlates with biopsychosocial outcomes. Amounts of exercise are primarily intended on improving pain and disability outcomes. To accrue health benefits, convergent information is found in Exercise is Medicine advice and a review explaining exercise induced analgesia (Exercise is Medicine, 2019; Sluka et al., 2018). Adults are advised at least 150 minutes per week of moderate-intensity aerobic activity, 75 minutes of vigorous aerobic activity, or a combination of both together with flexibility exercise (Exercise is Medicine, 2019; Sluka et al., 2018). Due to the wide variety of exercise protocols used to treat CLBP, the exercise amount recommended is a minimum of 3-4 times per week, with a guideline base of 10-15 minutes per bout, aiming for 30-60 minutes per day (Fielding et al., 2017; Exercise is Medicine, 2019; Sluka et al., 2018). Advised frequency varies between 3-7 days a week for aerobic exercise (Exercise is Medicine, 2019). For strengthening, a minimum of two to five days a week is recommended (Freburger et al., 2009; Exercise is Medicine, 2019; Sluka et al., 2018). Intensity is not finite and can be based on pain symptoms when progressing in exercise. Duration of exercise in a systematic review of 43 RCT's of 72 exercise treatments stated exercise exceeding 20 hours over a six-week period assists in decreasing CLBP (Hayden et al., 2005a). There was no significant difference between four exercise sessions over two weeks, versus eight sessions over four weeks, in terms of pain reduction (Callaghan, 1994).

According to the above guidelines, approximately 39% of UK adults and approximately 26% of US adults are not active enough to benefit their health (British Heart Foundation, 2017b; American Health Rankings, 2018). In South Africa, approximately 46% of adults between the ages of 25-64 would be classified as classified as insufficiently active (Guthold et al., 2011; Micklesfield et al., 2013).

Exercise for CLBP is often led by physiotherapist supervision in scheduled physiotherapy visits. In a national survey of physiotherapists in Ireland seeking mainly to reduce pain associated with CLBP, the majority (64% of respondents, n= 179) provided between six and 10 physiotherapy visits (Liddle, Baxter and Gracey 2009). This frequency lies in the upper range of physiotherapy visits with the nine-week non-specific exercise regimen advised in previous NICE guidelines (National Collaborating Centre for Primary Care [UK], 2009). Current NICE guidance acknowledges the persistence of CLBP requires longer term management (National Institute for Health and Care Excellence, 2019). The optimal number of treatments involving exercise or supervision necessary to achieve optimal outcomes for CLBP, has yet to be described.

Further research is required into whether walking as exercise, how much, and the amount of supervision required, is beneficial for treating CLBP over and above levels of ambient PA. Whilst exercise has been shown as one of the most effective strategies for managing CLBP (Liddle, Baxter and Gracey 2004; Maher, 2004; Rainville et al., 2004; Hayden et al., 2005b; van Tulder et al., 2006a;

Chou et al., 2007), the evidence for walking as an exercise for CLBP is controversial. A recent review examining whether walking was an effective treatment for CLBP, found it no more effective than other non-pharmacological treatment (Lawford, Walters and Ferrar, 2015). If the following were also measured: psychosocial components of kinesiophobia and catastrophizing, and objective measures of PA and walking delivered separately as an exercise, this would potentially improve practitioner delivery of evidence-based specific CLBP walking exercise and assist patient centred care.

Objectively measuring physical activity, exercise, and walking

Objective measures of PA can include step counts (Ainsworth et al., 2000; Tudor-Locke et al., 2011; Parker et al., 2017; Althoff et al., 2017). Step counts may vary by instrument used, placement of the pedometer, calibration, axis of measurement (i.e., bi, tri axial). A recommended target for healthy individuals is 10,000 steps per day (American College of Sports Medicine, 2011; Tudor-Locke et al., 2011).

Pedometers can be used to enable researchers, clinicians, and patients to better monitor walking activities. Pedometers and structured walking programs have been shown to help patients increase levels of PA (Merom et al., 2007; Baker et al., 2008; Kang et al, 2009; Harris et al., 2015). A systematic review of using pedometers to increase PA, included 26 studies with 2767 participants in total (Bravata et al., 2007). The systematic review included 8 RCTs where pedometer users significantly increased their PA by 2491 steps per day more than control participants (95% confidence interval [CI], 1098-3885 steps per day, $P < .001$). Pedometer users increased their PA by 26.9% over baseline, indicating that pedometer use is associated with significant increases in PA. There is some research that supports their use for motivating participants to be physically active though daily step count goals (Krein et al., 2013; McDonough et al., 2013; Harris et al., 2015). Two systematic reviews of dietary studies concurred that motivation to increase PA is significantly associated with pedometer use (Greaves et al., 2011; Dombrowski et al., 2012).

Pedometers often provide the user with visual feedback on the number of steps taken, can be inexpensive, unsophisticated, and usually measure in one axis only (vertical displacement). The UK National Institute for Clinical Excellence (NICE) supports pedometer use for research and PA encouragement (Harris et al., 2015). In a systematic review examining walking as a treatment for CLBP, pedometers were used in two of seven RCT's measuring walking to treat CLBP (Lawford, Walters and Ferrar 2015; Krein et al., 2013; McDonough et al., 2013). Pedometers are produced by several manufacturers. In a review of 837 articles analysing step counts in various populations, Omron pedometers were reviewed and assessed as suitable tools with which to measure walking in research (Tudor-Locke et al., 2011). In a randomised clinical trial by Rodriguez-Sanchez et al., (2014), the validated Omron Walking Style One 2.1, Hj-321 digital pedometer was used to assess a

three-month PA program amongst patients with dementia and their caregivers. The fundamental benefits of this pedometer were that it was simple and easy to use, had a digital clock, only monitoring steps taken and distance walked (Rodriguez-Sanchez et al., 2014).

As with pedometers, accelerometers are also available to objectively measure step counts. Accelerometers, on the other hand, tend to be more sophisticated, often working in more than one axis (vertical, sagittal, and horizontal). They tend to consist of a 'closed box', requiring connection to a computer and use specific software, to translate activity 'counts' into steps. The participant tends not to have the same visual display interaction found in a pedometer (Corder et al., 2007).

Pedometer-based step count ranges that classify the level of PA were developed and are described in Table 16 (Tudor-Locke and Bassett (2004)). People not achieving PA guidelines are recently referred to as insufficiently active, since people can be both sedentary and active (Thivel et al., 2018).

Table 16: Activity level defined by number of pedometer-based steps (Tudor-Locke and Bassett, 2004)

Number of daily steps taken	Activity level
0-5000	Sedentary
5000-7499	Low active
7500-9999	Somewhat active
10000-12500	Active
12500 or more	Highly active

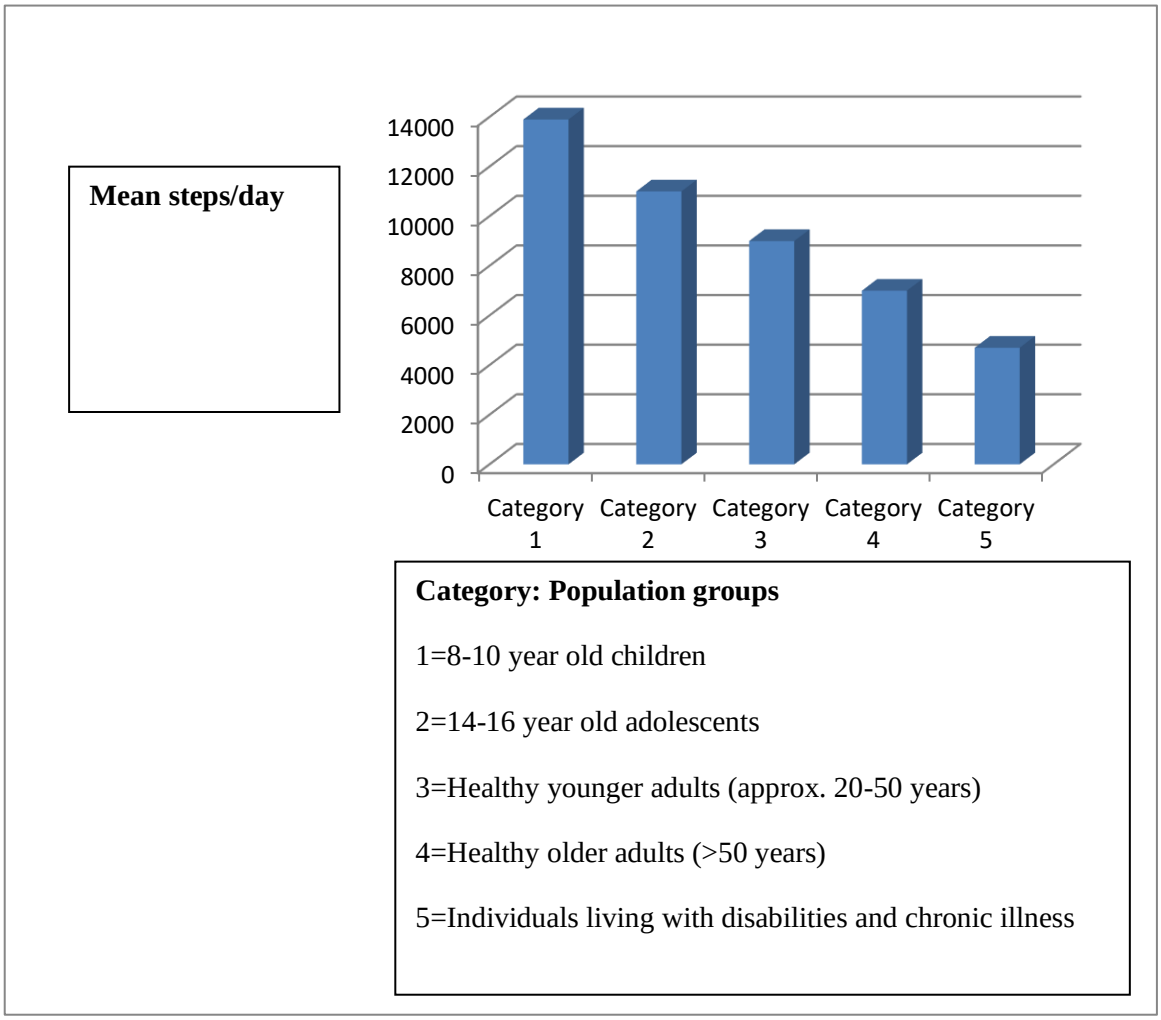
According to Tudor-Locke (2011), few adults achieve the 10,000-step target (Table 17). This has also been confirmed by other studies (Tudor-Locke, Hart and Washington 2009; Hirvensalo et al., 2011; Pillay et al., 2015; Parker et al., 2017; Althoff et al., 2017). The main reasons and predictors for not achieving these step counts include chronic pain (Parker et al., 2017), increased body fat (Pillay et al., 2015), increased age (>50 years) (Tudor-Locke, Hart and Washington 2009; Tudor-Locke et al., 2011), and cultural factors (Tudor-Locke et al., 2011; Hirvensalo et al., 2011). Mean daily step counts from both UK and South African populations, classify populations in Table 17 as being insufficiently active (Clemes et al., 2011; Althoff et al., 2017).

Table 17: Values of average daily step counts seen in different populations living without a disability or chronic illness.

Country	Average steps taken per day	Age range in years	Reference
United States of America	5100 (pedometer) 6500 (accelerometer)	18<	Tudor-Locke et al., 2011
Japan	7200 (pedometer)	15<	
Western Australia	9600 (pedometer)	18<	
Belgium	9600 (pedometer)	27-75	
United Kingdom	5500 (smartphone)	0<	Althoff et al., 2017
South Africa	4100 (smartphone)	0<	

Further data were collected demonstrating daily step counts below 10,000 in South Africa. These are observed in a cross-sectional study that used pedometers in a healthy population (Pillay et al., 2015). Using a valid and reliable Omron HJ 720 ITC pedometer, the study was completed by 312 participants (mean age 37±9 years). The mean steps/day in adult men and women was 7476 steps/day and 5769 steps/day respectively (Pillay et al., 2015). These step counts categorize this South African population of healthy individuals between individuals living with disability or chronic illness and healthy older adults over 50 years as determined by a systematic review of 32 studies examining step count to objectively quantify ambulatory activity (Tudor-Locke & Myers, 2001b) [Figure 6).

Figure 6: Expected steps/day for different populations. Adapted from Tudor-Locke and Myers (2001b).



Ten thousand steps are suggested as a reasonable daily step count for healthy adults (Yamanouchi et al., 1995; Hatano 1993; Tudor-Locke and Myers 2001b). However, empirical evidence supports the relation to cardiovascular and diabetes health-related outcomes (Tudor-Locke et al., 2002b; Bassett et al., 2003). Suggested values of steps per day can serve as benchmarks for comparison purposes but should not be misinterpreted as recommendations for appropriate activity levels among all illnesses. There is a paucity of evidence on how many steps are useful in treating appropriate outcomes related to CLBP. Recommendations can only be made once accumulated evidence supports specific health-related cut points (Tudor-Locke et al., 2002a). Researchers and practitioners require practice guidelines including step counts associated with health-related outcomes (Tudor-Locke & Myers, 2001b). No cut points in step count for achieving reduced pain, disability, kinesiophobia and pain catastrophizing for CLBP exist.

Subsets of the population may have different daily step counts, which may be affected by their physical or social status. In a study of 1,921 adults' aged 18 years and older an Accusplit AE120 pedometers was used to measure step counts (Bassett et al., 2010). Data were weighted to reflect the general U.S. population according to age, gender, race, education, and income. The results described that being male, of ages between 19-29 years old, having greater educational attainment, BMI classified as not overweight, and being single, were all positively associated with a higher number of daily step counts (Bassett et al., 2010) [Table 18]. Diversity within classifications of pain phenotypes, levels of disability, kinesiophobia and pain catastrophizing may also be seen if step counts are explored in populations with CLBP.

Table 18: Descriptive characteristics of pedometer-measured physical activity in the United States (Bassett et al., 2010).

Variable		Mean steps per day	p value
Gender	Male	5340	0.034
	Female	4912	
Age	19-29	5843	<0.001
	30-39	5127	
	40-49	5915	
	50-59	4742	
	60+	4027	
Education level	Less than high school	3920	0.059
	High school graduate	4947	
	Some college	5274	
	College graduate	5241	
	Some postgraduate study	5385	
Marital status	Single	6076	<0.001
	Married/ partner	4793	
	Divorced	5463	
	Widowed	3394	
BMI (calculated from self-reported height and weight)	Non-overweight/obese	5864	<0.001
	Overweight	5200	
	Obese	4330	

Experts from University College Hospital in London and the United Sates argue the earlier guidelines regarding 10,000 steps per day are 'hard to meet and are discouraging', supporting additional strategies to increase PA (British Heart Foundation, 2017a; Tuso, 2015). Insufficiently active individuals – i.e., those defined as people taking less than 10, 000 steps per day and people living with a chronic disease are estimated to take 3500-5500 steps/day (Thivel et al., 2018; Tudor-Locke

and Basset, 2004). This may suggest the 10,000 steps/day goal is unsustainable for particular populations. Setting these targets for CLBP treatment may risk attrition or failure.

In summary, a recent systematic review demonstrates that increases in PA are important in achieving health outcomes, however benefits are observed with smaller amounts than the 150 minutes minimum or 10,000 steps frequently recommended (Warburton and Bredin, 2017). This concurs with the statement that there is no universal goal existing for all populations (Tudor-Locke et al, 2002a). This seems reasonable in a CLBP context as unexplored step count recommendation may increase patients' pain and disability due to neurophysiological processes of increasing sensitization. Indeed, it is not yet known how many daily steps are ideal to treat CLBP. However, currently one study protocol is in the process of attempting to accumulate data for recommendations of step counts for a CLBP walking program over 12-weeks in the Saskatchewan province in Canada (Milosavljevic et al., 2015). Its methods reported using <7500 steps (inclusion criteria) per day as a cut off for interventions relative to health improvements, based on previous work (Tudor-Locke et al, 2008). It will be of interest what percentage of increased steps above the 7500 steps per day cut-off will demonstrate a significant change in Modified Oswestry Low Back Pain Disability Questionnaire (ODQ), International Physical Activity Questionnaire short form (IPAQ), Fear-Avoidance Beliefs Questionnaire (FABQ), and EuroQol health survey instrument (EQ-5D-5 L) outcomes used in the CLBP Canadian study. Furthermore, step counts associated with improved outcomes may add useable data to future CLBP exercise and walking interventions.

Two recent reviews state that walking is no more effective than usual care, strength specific exercise, medical exercise therapy and supervised exercise classes to improve pain and disability in adults with CLBP (Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). Gaps are noted in the reviews calling for more objective data on participant's PA at baseline, during and post intervention (Lawford, Walters and Ferrar 2015). Other gaps are to include insufficiently active participants to see what effects walking may have on their step count behaviour and outcome measures. It may be necessary to examine step counts of all treatment groups within a single study and not only those participating in walking interventions as noted in previous reviews on walking to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018).

Walking as treatment for chronic lower back pain

Three reviews have been done on the effect of walking on CLBP and shown it to be a useful adjunct treatment of pain and disability associated with CLBP despite acknowledging future research is required (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018). The main outcomes used were to examine pain intensity reduction, improved disability, return to work, and improved quality of life. Psychosocial outcomes have not been widely explored in these

reviews with only two latter reviews including them, focussing on kinesiophobia. In reviewing seven RCTs, Lawford, Walters and Ferrar (2015) found no evidence that walking is more effective than usual care, specific strength exercise, supervised exercise class or medical exercise therapy in improving quality of life and reducing disability in adults with CLBP. This was corroborated by a later review of nine studies highlighting though that walking should be advocated as a treatment for CLBP pain and disability (Sitthipornvorakul et al 2018).

Over time objective step measurements have become more detailed in the reviews however more step data within and between treatment groups may be necessary for future recommendations. Objective measurement of steps, using pedometers or accelerometery, was not done in the comparator groups to the walking interventions in any of the studies used in the three reviews (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018). Walking exercise duration, intensity and frequency is not consistent throughout the literature. One way of managing these factors is to base a walking program on evidence-based guidelines (American College of Sports Medicine, 2011). In the three reviews, when providing exercise established on evidence-based guidelines, or measuring PA changes objectively within the cohort, it appears to have significant benefit to pain and disability outcomes. In objectively supplying duration, intensity and/ or frequency changes in PA through steps taken, there may be an effect on CLBP outcomes notwithstanding pain, disability, and kinesiophobia. A recent paper which summarizes effects of PA in modulating chronic pain, concluded rather that insufficient activity be reduced rather than PA increased (Law and Sluka et al., 2017). Furthermore, the use of walking as exercise may have greater effect when used with insufficiently active populations, and this could be demonstrated if objective measurements were used from baseline until completion. Without further objective measurement of PA, or an example of an ideal amount of walking, the questions of suitability of walking as treatment for CLBP remain.

Table 19 includes only RCTs done with LBP that were defined as chronic, or pain lasting for more than three months (Hendrick et al., 2010; Lawford, Walters and Ferrar 2015; Sitthipornvorakul et al 2018). Methodological differences between the CLBP studies are described in Table 19. Case studies, cross-sectional studies, and studies with acute and subacute LBP with physiotherapy and walking were not included in the review since findings may not be comparable to RCT's on CLBP. With the paucity of studies done on walking to treat CLBP, and the variety in methodologies of walking exercise protocols used, implies best practice or definitive objective amounts of walking for optimal effects on outcome measures have yet to be discovered. However, there were key limitations in some of these studies that may have affected the outcomes. A fundamental limitation in a key RCT comparing walking to both exercise and usual care physiotherapy was the lack of a pragmatic control group, which implied improvements were attributed to the mean (Hurley et al., 2015). A limitation

for some studies included small sample size which reduced the power to detect differences between baseline and follow-up (Eadie et al., 2013; Karadeniz et al., 2014; McDonough et al., 2013). Sample sizes ranged from n=60, n=18, n=56 respectively. Being insufficiently active were not inclusion criteria in several studies hence different levels of PA in participants may affect their response to advised walking interventions (Torstensen et al., 1998; Koldaş Doğan et al 2008; Eadie et al., 2013; Karadinez et al., 2014; Magalhães et al 2015; Cho et al., 2015). Baseline PA levels were not monitored using the same measure between studies, nor objective changes in PA throughout the walking interventions. This limited the objective measurement of the effect of walking on populations with the same level of baseline PA or observing changes in step count from baseline throughout the studies (Torstensen et al., 1998; Eadie et al., 2013; Shnayderman and Katz 2013; Karadinez et al., 2014; Magalhães et al 2015; Cho et al., 2015). Between and within treatment group step count has not yet been done, so comparison of steps between treatment types requires further examination (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018). A cohort of 229 veterans was recruited from one medical centre (Krein et al., 2013). Even though the primary outcome of sub-group analysis of moderate disability showed significant improvement ($p=0.01$) of the intervention group using a pedometer driven walking program and educational tools compared to control group, the trial produced results with low generalizability due to participants only recruited from one centre. Reduced treatment fidelity due to high number of treating therapists, and a lack of blinding in patients and therapists was observed in Hurley et al., (2015). A randomised clinical trial for CLBP compared supervised Nordic walking to unsupervised Nordic walking and advice to stay active (Hartvigsen et al 2010). However, inclusion criteria stated LBP for more than eight weeks which does not meet criteria for classifying CLBP which is defined by symptoms for more than three months.

Despite varied treatment methodologies used in combination with walking, the role of expectation of pain outcome changes may be an important and unexplored factor. A systematic review of studies from the United Kingdom, included studies that measured satisfaction post treatment; however, expectation of change in pain intensity was not measured (Lawford, Walters and Ferrar 2015). An article discussing neurological underpinnings of pain, the role of placebo, clinical applications, and the context of society, concludes that patients experience pain at a level influenced by their expectation (Tracey 2016). This article highlighted the importance of measuring expectations of pain outcomes in clinical contexts. This was further corroborated by the need to evaluate health services from patient perspectives (Liddle et al., 2007; Sanderson et al., 2010; Blank and Burau, 2010; Froud et al., 2014). Some South African studies have acknowledged the use of patient's expectation in treatment (Westway et al., 2003; Narasimooloo, 2011). However, none of the reviews involving walking to treat CLBP had measured what expectation of improvement participants had before

beginning treatment (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018).

Outcome measures used when treating CLBP with walking.

Outcome measures that have frequently been used were pain and disability; however psychosocial outcomes have not been measured extensively in studies using walking to treat CLBP. Outcome measures that have and have not been used will be discussed below. Table 19 shows study details included in three reviews using walking to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015, Sitthipornvorakul et al 2018). Table 19 displays the key findings of pain intensity as this was the primary outcome measure in the current RCT.

Table 19: Walking study comparison used in three reviews on walking to treat CLBP

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Study design & author
2 groups- Both received vertical ambulatory traction for 12 days/ 20-minutes per session. Followed by 8 more sessions of 30 minutes on alt. days Control: Sit/stand no walking intervention Intervention: Walked an additional 15 minutes / session on a treadmill.	28 days (20 treatments) intervention Follow-ups completed at baseline, after completion ±1 month, 6 months, 12 months	Pain Visual Analogue Scale 0-10 demonstrated on a bar graph Range of movement: ROM Patient satisfaction	Intervention group had significant pain reduction ($p<0.0001$), increased ROM, increased satisfaction compared to the control group. Pain intensity at baseline for both groups <7/10, >6/10. Pain intensity at one-month: control = 4/10 intervention group >2/10 <3/10. Pain intensity at six months: control = <5/10 >4/10 intervention group 3/10. Pain intensity at 12 months: control <5/10 >4/10 intervention group <4/10 >3/10	Mechanical CLBP confirmed by X-Ray, CT or MRI scan (42 male, 34 female) Mean age 49.2 (control), 48.6 (intervention)	Randomised controlled trial: RCT (Mirovsky et al., 2006)
Interventions lasted 1 hour 3 times a week for 12 weeks. Conventional physiotherapy (CP): heat, cryotherapy, massage, electrotherapy, traction, unspecified exercise Medical exercise therapy (MET): Medical exercises in classes advised in sets, repetitions, ROM. 7-9 exercises with 1000 repetitions between them in total. Walking group/ self exercise (SE): Walk unsupervised 1 hour 3 times a week for 12 weeks	12-week intervention Follow-ups completed at baseline, 3 months, 12 months	Pain Visual Analogue Scale Oswestry Disability Index: ODI Sick Leave Patient satisfaction	CP & MET had statistically significant decreased LBP ($p=0.01$) and leg pain ($p=0.003$) versus the walking group. Baseline, Mean (S.D.); CP= 50.9 (19.2); MET=53.1 (21.3); SE= 55.0 (21.0). After 12 weeks of treatment, Mean (S.D.); CP= 39.0 (28.0); MET=37.2 (25.3); SE= 50.4 (27.2). CP & MET had statistical significant decreased ODI ($p=0.01$) versus the walking group. Satisfaction scores were Conventional physiotherapy (32.2%), medical exercise therapy (34.2%), walking group (9.5%).	208 CLBP patients (103 male, 105 female) Mean age 42.1 (medical exercise therapy), 43.0 (conventional physiotherapy), 39.9 (unsupervised walking)	Randomised controlled trial: RCT (Torstensen et al., 1998)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1: Supervised aerobic treadmill walking three times/ week for six-weeks. Walk for 40-50 minutes at 65%-75% max HR plus daily defined home exercise program (flexion, extension, mobilization and stretching). Group 2: Physiotherapy (hot packs, electrotherapy) plus defined home exercise program (flexion, extension, mobilization and stretching). Group 3: Daily defined home exercise program (flexion, extension, mobilization and stretching).	Group 1: Walking three times a week for six-weeks. Group 2: Physiotherapy three times a week for six-weeks Group 3: Daily Follow-ups completed at baseline, 6 weeks	Pain Visual Analogue Scale (0-100). Roland Morris Disability questionnaire: RMDQ Becks depression Inventory General Health questionnaire Exercise test duration Met scores Spinal mobility/ Schober test	No significant difference between groups in pain, disability and depression scores. VAS (mean, SD): Group 1 baseline 57.0±24.5, follow-up 34.1±27.6 p=0.002. Group2 baseline 61.2.3±20.5, follow-up 28.8±28.1 p=0.0001. Group 3 baseline 56.0±19.9, follow-up 33.6±24.3 p=0.001. Physiotherapy and home exercise showed largest improvements in disability and depression scores. General Health Questionnaire improved in groups 1&2. Exercise test duration and MET scores similar improvements in all three groups No significant difference was found between and within groups spinal mobility	CLBP patients started (15 male, 45 female). Mean age 40.2 years±8.4 years	Randomised controlled trial: RCT (Koldaş Doğan; et al., 2008)
Group 1: Supervised treadmill walking (3-3.5 km/h) for 30 minutes three times a week plus lower back exercises and ergonomic advice for 30 minutes three times a week of 14 exercises Group 2/ Control: Lower back exercises and ergonomic advice for 30 minutes three times a week of 14 exercises	8- weeks Follow-up completed at baseline and at 8 weeks	Pain Visual Analogue Scale (0-100) Oswestry Disability Index Lower Back Extensor strength	VAS and ODI trend improved in both groups. VAS (mean, SD): Group 1 baseline 31.3±17.9, post treatment 16.9±9.3. Group2 baseline 36.3±17.4, post treatment 20.5±13.1. Increased strength at 12°, 24, 36° of lumbar flexion in treadmill walking group.	CLBP (20 male patients, ten in each group). IVG: 27.7±4.2 years CG: 29.1±4.8 years	Randomised trial (Cho, Y.K., and Kim, D.Y., 2015)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1 Walking program up to 30 minutes 5 times/ week Group 2 Supervised exercises 1 times a week for 8 weeks Group 3 Usual care physiotherapy (exercise, manipulation and education at the physiotherapists discretion)	8-weeks intervention Follow-ups completed at 12 weeks and 6 months	Numerical rating scale (back and leg pain) Oswestry Disability Index: ODI Quality of life: SF-36 Mental component score: MCS Pittsburgh Sleep Quality Index Insomnia severity index International physical activity questionnaire Fear avoidance behaviour questionnaire: FABQ Anxiety Depression	All 3 groups improved with large CI's with the smallest seen in the walking group. Results presented as change in NRS and ODI scores presented as means ±95% confidence interval. At 12 weeks- Average LBP Group 1: -0.68(-2.10 to 0.75); Average leg pain: 0.50 (-0.73 to 1.73); Group 2: -0.68(-1.58 to 0.22); Average leg pain: 0.43 (-1.33 to 2.19); Group 3: -1.96 (-3.53 to 0.39); Average leg pain: -0.31 (-2.65 to 2.04). At 12 weeks- ODI: Group 1: -3.35 (-8.97 to 2.26); Group 2: -7.14 (-15.63 to 1.35); Group 3: -9.06 (-18.31 to 0.19)	CLBP (23 male, 37 female) Mean age 44.9 years	Randomised controlled trial: RCT (Eadie et al., 2013)
2 groups: walking three days/ week Group 1: Treadmill walking: 20-60 minutes supervised Group 2: Overground walking: unsupervised	4-week Intervention Completed at baseline, 4 weeks	VAS Pain scores Oswestry Disability Index: ODI Quality of life: SF-36 Spinal Mobility VO_2 Max, Max HR, MET, Anaerobic threshold.	G1: ODI&VAS improvement p=0.025 G2: ODI&VAS improvement p<0.025 G2: significantly greater decrease in ODI vs. G1 (p=0.025). VAS decrease similar between group (p>0.05). G2: showed a significant decrease in time to reach target HR (p=0.008). degree of change between G1&2 similar between group (p>0.05).	10 women 8 men CLBP without radicular pain. Mean age 56.4 years	Randomised controlled trial: RCT (Karadeniz et al., 2014)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1: Group exercise classes: once a week for 1 hour for 8 weeks (up to 10 exercises with 3 levels of difficulty, including: warm up, stretching, aerobic, trunk and upper and lower limb strengthening, cool down and relaxation) Group 2: Individualized walking program (variable 10-30 minutes 4-5 times a week) Group 3: Usual care physiotherapy: multimodal, education, advice, manipulation, exercise based on physiotherapists discretion) Expected visits based on a mean of 5 visits per 8 weeks.	8-weeks Intervention. Follow-ups from baseline outcomes at 3, 6, and 12 months	NRS Pain scores Oswestry Disability Index: ODI Fear Avoidance-Physical activity EuroQOL Weighted Health Index (EQ-5D) Back Beliefs questionnaire Exercise Self-efficacy questionnaire Weighted Health Index Readiness to change questionnaire Patient satisfaction	Significant improvements in NRS-average pain, ODI, fear avoidance for physical activity, exercise-self efficacy questionnaire between baseline and at 12 months follow-up. NRS (mean ±95% confidence interval): Group 1 baseline 5.65(5.23-6.06), 12 weeks 5.17 (4.71-5.62). Group 2 baseline 5.59(5.18-6.01), 12 weeks 4.78 (4.32-5.24). Group 3 baseline 5.77(5.36-6.19), 12 weeks 4.55 (4.10-5.01). Results presented as change in NRS presented as means ±95% confidence interval. At 12 weeks- Group 1: -0.43(0.07 to -0.94); Group 2: -0.97(-0.45 to -1.48); Group 3: -1.16(-0.66 to -1.66). No significant change between group in satisfaction with care or outcomes at three months (p.0.05). Satisfaction with care not reported at 12 months. EuroQOL Weighted Health Index improved but no significant changes over time. No significant change in Back Belief questionnaire at 12 months follow-up.	CLBP 79 men 167 women were randomised 219 received treatment Mean age 45.4 years	Randomised controlled trial: RCT (Hurley et al., 2015)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1/ intervention: Pedometer-based internet mediated walking program with goals and feedback Group 2/ Enhanced usual care: Participants to upload pedometer data	12-month intervention Follow-ups at 6 and 12 months	Roland Morris Disability Questionnaire: RMDQ MOS general pain and function measure NRS (0-10) Kinesiophobia Depressive symptoms Health related quality of life Pain intensity Daily steps	Internet mediated group (Group 1) reported less back pain disability (at 6 months but not at 12 months), increased physical activity and less pain (at 6 months). Group 1 NRS mean (SD): baseline 6.0 (SD not given), six months 4.7 (2.1). Group 2 NRS mean (SD): baseline 6.1 (SD not given), six months 5.2 (2.1). Adjusted between-group difference (95% CI) 0.5 (-0.01 to 0.98) was not significant p=0.06	Mean age 51 years 200 females 29 males n=229	Randomised controlled trial: RCT (Krein et al., 2013)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1/ Intervention: treadmill walking graded moderate using heart rate monitor, CLBP education using Back Book, strength exercises Group 2/ Physiotherapy group: Stretching, strengthening and motion control exercise	Each treatment was done for one hour, twice a week for six-weeks. Follow-ups at baseline and at six-weeks	NRS 0-10 Pain scores Roland Morris Disability Questionnaire: RMDQ McGill Pain Questionnaire Health related quality of life Global perceived effect Return to work Tampa Scale of Kinesiophobia: TSK Baecke Questionnaire: Daily habitual PA Physical Capacity	NRS (mean, SD): Group 1 baseline 7.2±2.1, post treatment 2.4±1.8 $p<0.001$. Group2 baseline 7.6±1.7, post treatment 2.6±1.6 $p<0.001$. All outcome measures improved for both treatment groups showed significant improvement ($p<0.05$) There were no statistically significant differences between groups Therefore, no form of exercise is superior to another for CLBP treatment.	IVG: 9 males 24 females mean age 47.2 (±10.5) Physiotherapy group: 8 males 25 mean age 46.6 (9.5)	Randomised controlled trial: RCT (Magalhães et al., 2015)

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group1/ SEG strength exercise group with stretching Group2/ CEG combined exercise group (strength exercise and walking exercise using a step box) with stretching Group 3/ CG control group	Each treatment group performed supervised exercise for 50 minutes twice per week for 12 weeks. Follow-ups completed at baseline, 12 weeks	VAS Pain score (0-100) Back flexibility Back strength Roland Morris Disability Questionnaire: RMDQ Muscle mass change Fat mass change	VAS (mean, SD): Group 1 baseline 32.3±14.9, post treatment 22.0±1.3. Group2 baseline 45.3±14.8, post treatment 33.1±20.0. VAS showed significant group differences (p=0.04) Flexibility showed no significant changes Back strength significant difference over time but group differences were not significant Post hoc analysis showed significant differences between SEG and CG, and CEG and CG with no difference between exercise groups. RMDQ showed significant time interaction with group (p<0.04). 12 weeks of combined exercise can demonstrate decreased disability Muscle mass showed no significant difference Fat mass showed significant group difference (p=0.04). Time interaction with group was significant (p<0.04) between SEG and CG demonstrating fat mass reduction using strength exercise programs.	CLBP (all overweight (BMI>23/kgm²) SEG n=15 mean age 42.7±13.4 CEG n=15 mean age 46.7±8.1 CG n=6 mean age 43.3±9.9	Randomised controlled trial: RCT Lee and Kang 2011

Interventions / experimental conditions	Length of intervention	Outcome measures	Findings of the study	Population group	Type of study & author
Group 1: Pedometer driven walking program and advice to stay active with education n=39 Group 2: Advice to stay active with education n=17	8-week intervention, follow-ups at 9 weeks and 6 months	NRS Pain scores Oswestry Disability Questionnaire Physical activity Fear avoidance behaviour questionnaire: FABQ Quality of life: EQ5D Self-Efficacy Back Beliefs Outcome State of change Patient Satisfaction	Larger mean improvement in pain (d=0.4) and physical activity (d=0.59) in the walking program group. Results presented as mean unadjusted change in NRS (±95% confidence interval) and between group effect sizes. At 9 weeks- Group 1: -0.9(-1.6 to -0.1); Group 2: -0.7(1.6 to 0.2) between group effect sizes -0.1; At 6 months- Group 1: -1.6(-2.6 to -0.6); Group 2: -0.5(-1.8 to 0.8) between group effect sizes -0.4. The walking group also demonstrated on Oswestry Disability Questionnaire 8.2%-point improvement (95% CI, -13 to-3.4) compared to 1.6% (95% CI, -9.3 to 6.1) on the advice/ exercise only group. Fear avoidance and self-efficacy had small improvements over time with small differences between groups.	CLBP Mean age 49.5 years 25men 31 women	Randomised controlled trial: RCT (McDonough et al., 2013)
Both interventions twice a week for six-weeks Group 1: Moderate intense treadmill walking Group 2: Specific low back exercise	6-week intervention	Fear avoidance behaviour questionnaire: FABQ 6-minute walk test Back and abdomen muscle endurance tests Oswestry Disability Index: ODI Lower back Function: LBPFS	Significant improvements in all outcomes in both groups, non-significant differences between groups. Pain intensity was not used therefore analysed.	CLBP Mean age 45.3 years 11 males 41 females	Randomised controlled trial: RCT (Shnayderman and Katz-Leurer, 2013)

Pain

Pain was measured as an outcome in the three reviews (Table 19). When walking and additional treatment modalities were combined in studies in the three systematic reviews, it is unknown which factors were responsible for pain reduction. Many factors involved in modalities added to walking were manual therapy and/or education, and/or additional strength/stretching/stabilization exercises. Results show this may have positively influenced the pain outcomes. The trials included in Table 19 have varied results and differences between studies worth noting in statistical significance and clinical significance when examining pain intensity. When comparing two trials with similar treatment groups these differences are noticeable. In a fully powered RCT where all interventions were combined with education, walking was compared to usual care physiotherapy and exercise classes, there was no statistically significant differences between the three groups at follow-up ($p=0.476$) (Hurley et al., 2015). Although in the same trial no MCID in mean pain intensity scores ($\geq 2/10$) were noted between treatment groups, the largest percentage of MCIDs was observed in the walking intervention. Divergent information was observed in an earlier RCT with similar treatment groups. Walking was used as a treatment with no addition of manual therapy, education, or strength/ stretching stabilization exercises and demonstrated no statistically significant change in pain outcomes in the walking group (Torstensen et al., 1998). Pain after 12 weeks of treatment when compared with pre-treatment showed a highly significant difference ($p=0.00006$) in favour of the medical exercise therapy and conservative physiotherapy groups versus the walking treatment group (Torstensen et al., 1998). However, none of the groups reported a minimally clinically important difference (MCID) in pain intensity ($\geq 20\%$) on a VAS after 12 weeks of treatment, although the difference in pain intensity reduction was the smallest in the walking group (Torstensen et al., 1998). The remaining studies which used walking in combination with another modality all showed a significant improvement in pain intensity which is noted in Table 19 (Mirovsky et al., 2006; Koldaş Doğan et al 2008; Eadie et al., 2013; McDonough et al., 2013; Karadeniz et al., 2014; Hurley et al., 2015 Magalhães et al 2015; Cho et al., 2015; Lee and Kang 2016).

A MCID is indicated by greater than 20 percent improvement pain intensity score (Farrar et al., 2001; Hägg, Fritzell and Nordwall, 2003; Ostelo and de Wet, 2005; Haefeli and Elfering, 2006, Suzuki et al., 2020). Only three studies in Table 19 demonstrate this difference in mean pain intensity scores. Only one of these studies showed a statistically significant difference between pain scores at all follow-up points in favour of the walking and traction ($p<0.0001$) (Mirovsky et al., 2006). The remaining two studies showed no statistically significant difference between treatment groups (Koldaş Doğan et al 2008; Magalhães et al., 2015). A six-week RCT comparing walking and home exercises to physiotherapy modalities and also to home exercises showed a MCID greater than 20% in all treatment groups (Koldaş Doğan et al 2008). The greatest MCID (greater than 30%) was seen in the physiotherapy group. A six-week RCT comparing a walking program, education on CLBP and

strength exercises to a treatment group using physiotherapy exercises displayed MCIDs over 40% in both groups (Magalhães et al., 2015). Only the intervention group using walking and ambulatory traction in the third study showed a MCID of more than 20% (Mirovsky et al., 2006). Between the three studies, the intervention length was six weeks or less and positive MCID may have been due to recording pain intensity in this period. RCTs in Table 19 with follow-ups eight weeks or longer reported smaller improvements in mean pain intensity score. The ambulatory traction trial however is the only trial of its kind comparing manual traction alone to walking combined with manual traction. The suggestion of walking exercise added to a manual therapy may have clinical and statistically significant changes regarding pain intensity.

Manual therapy was combined with walking in two studies, both showing reductions in pain intensity (Mirovsky et al., 2006; Karadeniz et al., 2014). A RCT investigating the effects of walking combined with vertical ambulatory traction compared with vertical ambulatory traction alone, found that pain had reduced significantly ($p<0.001$) more in the group with walking exercise addition (Mirovsky et al., 2006). Both treatment groups displayed reduced pain on a VAS bar graph, however only the group with walking exercise added displayed a MCID of >2/10 at all follow-up time points. This early study suggests adding manual therapy using a traction device to walking may enhance clinical improvements in pain intensity. A RCT using heart rate monitors to track pacing comparing treadmill walking to over ground walking found pain reductions in both groups (Karadeniz et al., 2014), however manual therapy factors were added to walking treatments. Both groups had physiotherapy five times per week and hydrotherapy twice a week for four weeks. Interactions of factors involved in physiotherapy and hydrotherapy may have positively influenced the pain outcomes. Walking may not solely have been responsible for reduced pain outcomes. Mean pain scores were not discussed in the results (Karadeniz et al., 2014). It is unknown if a MCID was achieved in this four-week intervention using walking and physiotherapy combined.

Delivery of education about CLBP when combined with walking treatments may influence pain outcomes. Walking exercise was compared to both usual care physiotherapy and medical exercise therapy for CLBP in Torstensen et al., (1998); Eadie et al., (2013) and Hurley et al., (2015). Standardized education via the ‘Back Book’ on LBP was not included in the methodology of one of these trials, and significant reductions in pain intensity were not observed in the treatment group using walking (Torstensen et al., 1998). Accordingly, one study used walking combined with CLBP education compared to education alone (McDonough et al., 2013). The education was based on the Back Book, and both treatment groups showed reductions in pain intensity (McDonough et al., 2013). However, in a RCT a treatment using walking, strength training, and education from the Back Book was compared to physiotherapy with no education and pain improved in both groups significantly (Magalhães et al 2015). The significant improvement in pain in the physiotherapy group may be in

response to factors other than education, for example the process of caring for the patient through physiotherapy attention. The requirement of education on CLBP delivery requires more attention as results show varied methods of CLBP education application, if any. Pain reduction was seen in both treatment groups where education combined with both treatment groups was not based on pain management as seen in the Back Book but on mechanical ergonomic principles (Cho et al., 2015). Two studies seen in Table 19 had no education on CLBP in any intervention, and all interventions demonstrated a significant reduction in pain intensity (Koldaş Doğan et al 2008; Lee and Kang 2016). The earlier review on using walking to treat CLBP may indicate that CLBP education and advice in addition to treatment or used independently influence pain reduction (Lawford, Walters and Ferrar, 2015). However, studies involved in the latter review indicate that even treatments which do not involve education have a pain-relieving effect (Sitthipornvorakul et al., 2018). These differences in treatment delivery may be worth exploration if education on CLBP is to be used to facilitate greater pain reduction than without education.

Evidence-based walking programs may have led to reduction in pain intensity. Three studies showing pain reduction in groups using walking as treatment used evidence-based walking programs. Two were based on ACSM guidelines (Eadie et al., 2013; Hurley et al., 2015). Another was based on the 5 A's framework (McDonough et al., 2013). Comparatively, walking used in Torstensen et al., (1998) was not based on an evidence-based guideline and was the only walking treatment not to show significant reductions in pain intensity when compared to usual care physiotherapy and medical exercise therapy for CLBP. However, in the latter review, trials which used walking not based on any evidence-based walking programs had reductions in pain when compared to various other CLBP exercise programs (Koldaş Doğan et al 2008; Magalhães et al 2015; Cho et al., 2015; Lee and Kang 2016). These studies recognize that walking was potentially beneficial to pain outcomes as PA is considered beneficial, however, walking structured on evidence-based guidelines, typically has frequency, intensity and/ or duration that can be progressively manipulated was not used. This highlights that potentially an ideal walking program to treat CLBP pain outcomes has not yet been formulated.

Objective monitoring of step counts via pedometer or accelerometer may be beneficial understanding reductions in pain intensity. Differences in step counts between treatment groups may affect understanding pain outcomes. No single study using active treatments has thus far objectively measured steps in all treatment groups (Table 19). Furthermore, only two studies showed increased objectively measured step counts from baseline to completion in the walking group (Krein et al., 2013; McDonough et al., 2013). The benefit of only providing intervention groups doing walking is that measuring steps using a pedometer is known to motivate increased PA (Merom et al., 2007; Baker et al., 2008; Kang et al, 2009; Greaves et al., 2011; Dombrowski et al., 2012; Harris et al.,

2015). If treatment groups not randomized to walking measured their steps, participants may have extrinsically been motivated to increase PA. The disadvantage however is not being able to compare PA through steps between and within these groups in the same study. Four studies seen in Table 19 employed objective measures in groups using walking as a treatment (Krein et al., 2013; Eadie et al., 2013; McDonough et al., 2013; and Hurley et al., 2015). However, lack of PA data and low compliance of accelerometer wear time was noted as a limitation (Eadie et al., 2013; Hurley et al., 2015). In the latter review walking was not measured using objective pedometery or accelerometery programs (Koldaş Doğan et al 2008; Magalhães et al 2015; Cho et al., 2015; Lee and Kang, 2016). Therein, the objective effect of number of steps taken on pain outcomes is unknown in these studies.

Insufficiently active and active populations may respond differently to treatments used in CLBP studies using walking as a treatment. The impact of PA on improving fitness, strength and feelings of wellbeing associated to increased PA in insufficiently active populations is documented (Law and Sluka et al., 2017; Warburton and Bredin, 2017). The benefit of added PA to insufficiently active or active populations is equivocal in pain outcomes in studies using walking as a treatment. Furthermore, in an earlier review on walking to treat CLBP, one study population did not specify the inclusion of only insufficiently active individuals (Lawford, Walters and Ferrar, 2015; Torstensen et al., 1998). Later studies using walking as a comparative treatment included only insufficiently active participants (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). Inclusion of insufficiently active individuals in three RCT's may have been the reason walking as a treatment showed reduced pain outcomes, since participants which may have been physically active may not have responded exponentially to an exercise intervention. Only some of the studies employed objective measures via accelerometery prior to baseline to include insufficiently active populations (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). However, significant pain reductions were seen in all interventions in the recent review (Sitthipornvorakul et al., 2018). Only one study in the review described a subjective measure of insufficiently active at inclusion (Lee and Kang, 2016). The other CLBP studies in the recent review did not specify inclusion of insufficiently active participants (Koldaş Doğan et al 2008; Magalhães et al 2015; Cho et al., 2015). When examining the effects of walking as a treatment on pain intensity, comparing the effects on insufficiently active to active populations may be worth exploring in order to understand what effects walking has on CLBP populations with varied levels of PA.

Supervised exercise programs are most likely to reduce pain outcomes in CLBP (Hayden et al., 2015a). Varied levels of supervision were apparent in treatment groups in three reviews (Table 19). Supervision was provided in each treatment provided by a clinician present during the treatment, in a group exercise, via the telephone or the internet. It is unclear if mode of supervision was associated with pain reduction. It is a key limitation that the supervision or number of visits was not examined

as an independent variable associated with pain intensity in any of the studies in table 19. The effect of participant supervision may require further exploration in CLBP studies outcomes including pain intensity. Recording participant's pain during treatment may modify clinical supervision depending on whether pain symptoms change with allocated treatment. Internet log ins were used to maintain supervision in one walking study (Krein et al., 2013). Paper diaries were used in another walking study to record adverse events and walking program (McDonough et al., 2013). No physiotherapy studies using walking incorporated diaries to monitor pain. Decreasing the need for interviews, patient diaries used on participants with pain of the age range 18-65 years were an alternative to capturing patient perceptions of their pain (Miller, Pinnington and Stanley, 1999). Participants need not rely on recall and can demonstrate an up-to-date daily experience of their pain typified by diary use in complementary scenarios in pain clinics (Follick, Ahern and Laser-Wolston, 1984). Diaries can have limitations. In one pain study, diaries were qualitative and excluded participants with a lack of basic literacy skill and included participants of the age range 18-65 years. Furthermore, the crude response rate using diaries was 40% (Miller, Pinnington and Stanley (1999). Advantageously however, evening pain score entries were more frequent and reportedly more convenient compared to morning entries (Miller, Pinnington and Stanley, 1999). Pain diaries may provide supervisory information to researchers and clinicians alike.

Disability

Measuring disability experienced by patients with CLBP is critical to function and quality of life. Disability was measured using either the Oswestry Disability Index (ODI) or Roland Morris Disability Questionnaire (RMDQ) in several studies (Table 19).

Reduced disability is seen at follow-up in varied comparators in studies using walking to treat CLBP. Varied belief in a treatment for CLBP between population groups may affect disability outcomes, including other factors such as whether the walking is performed on a treadmill or outdoors.

Differences in population's belief in treatment may affect disability outcomes. A Turkish population showed statistically significant improvement in disability when exposed to physiotherapy and home exercise ($p=0.01$) unlike exposure to walking and home exercise (Koldaş Doğan et al 2008). The authors stated that this positive outcome may be related to stronger population belief in physiotherapy treatment using hot packs and electrotherapy rather than walking (Koldaş Doğan et al 2008). Two trials conducted in Ireland showed no statistically significant difference between groups in the change in disability scores (Eadie et al., 2013; Hurley et al., 2015). The two RCT's compared the effectiveness of physiotherapy using three groups: a walking program, supervised exercise, and usual care physiotherapy on CLBP (Eadie et al., 2013; Hurley et al., 2015). When observing these different results, two geographically similar Irish populations may have been historically exposed to a

different context and content of CLBP treatment to Turkish patients. These differences suggest the study of participant expectation of treatment outcome is worthwhile.

When over ground walking was compared to walking done a treadmill to treat CLBP, there were significant reductions in the former treatment group (p=0.02) (Karadeniz et al., 2014). The authors propose the influence of well-being associated to the outdoors reduced disability; however, this association was not investigated further in the trial.

Observing step count affecting disability related to walking interventions was well documented in one trial. A feasibility trial comparing pedometer driven walking and CLBP advice to CLBP advice alone, summarized that patients who increased their PA through increased steps demonstrated reduced CLBP associated disability (McDonough et al., 2013). At six months the group with a walking intervention demonstrated a mean improvement in the Oswestry Disability Questionnaire of 8.2% points (95% CI, -13 to -3.4) compared to 1.6% (95% CI, -9.3 to 6.1). At baseline, all 57 Irish participants were issued with ActivPAL accelerometers which confirmed that both groups included had an objective measure of insufficiently active baseline PA. This demonstrated the validity of the results using interventions on participants with CLBP that are insufficiently active. The group using walking and education as a treatment wore pedometers and results showed an increase in mean steps per day taken from baseline to follow-up (2776 steps). Unlike previous RCT's this was an improvement in understanding whether increased steps on an insufficiently active CLBP population positively influences disability outcomes. The baseline accelerometer data ensured objective inclusion of insufficiently active participants unlike comparative studies using walking to treat CLBP. Although only the group using walking used pedometers to further measure steps throughout the trial. Objectively measured steps between the two groups could not be compared nor could it be determined if steps correlated with disability outcomes in both groups.

A RCT investigated the effect of an aerobic treadmill walking program versus a lower back muscle strengthening program on disability and fear avoidance in CLBP participants and showed significant improvements in disability in both groups (Shnayderman and Katz-Leurer, 2013). Both groups were treated twice a week for 6 weeks, with no significant difference between groups (Shnayderman and Katz-Leurer, 2013). Neither group used objective measurement comparing walking, PA or exercise done during the trial. Only a six-minute walk test was compared between groups from baseline to follow-up as an objective measure. The authors also concluded that findings may have been more illustrative if subgroups based on CLBP classification according to signs and symptoms were first identified. This subgrouping for modelling purposes using pain phenotypes may hold for any study using walking to treat CLBP disability which no study had yet done.

The review by Lawford, Walters and Ferrar (2015) states that walking studies with heterogeneous methodologies demonstrate that walking is no more effective in improving disability than strengthening, progressively graded medical exercise, and supervised exercise classes; with the limitation that walking had not been compared to a true control (no intervention).

Psychosocial Outcome Measures

When comparing three reviews (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018), only the latter two reviews highlighted the use of biopsychosocial outcomes as secondary outcomes. The biopsychosocial outcome included frequently was fear avoidance, but pain catastrophizing was not included at all. Six studies in the latter reviews supported the use of measuring fear avoidance as a contributing variable to CLBP (Shnayderman and Katz-Leurer, 2013; Eadie et al., 2013; McDonough et al., 2013; Karadeniz et al., 2014; Hurley et al., 2015; Magalhães et al 2015). When comparing a walking program, supervised exercise, and usual care physiotherapy, two RCTs using the same interventions demonstrated a small mean reduction of fear avoidance behaviour in all groups (Eadie et al., 2013; Hurley et al., 2015). A significantly greater reduction was noted, in the walking program, with those who achieved clinically significantly improvements in pain (NRS) and disability (ODI) suggesting the mediating effect for fear avoidance on pain and disability (Hurley et al., 2015). The study population included was insufficiently active. The exposure to increased PA in the walking group may indicate greater reductions in fear avoidance when using walking in CLBP patients. Similarly, when comparing walking and CLBP advice to advice alone, small improvements in fear avoidance at follow-up were found in both insufficiently active groups in favour of the group using walking (McDonough et al., 2013). Changes were similar between groups and regarded as unlikely to be clinically important due to the small change. The improvements in three trials were likely to be result of re-assurance through advice, mitigating fear avoidant behaviour, which was common to all (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). A limitation noted in one RCT was that the mean baseline score for fear avoidance was low suggesting a selection bias of participants agreeing to participate due to low fear of movement (Shnayderman and Katz-Leurer, 2013). Other studies using walking for chronic pain, not specifically CLBP, did use catastrophizing as an outcome to successfully rehabilitate patients (Sullivan et al., 2006c). Using walking as a treatment may utilize the Beckian model (Beck, 1995). Identifiable automatic maladaptive cognitions could be replaced by rational thinking though demonstrating new behaviour by adding PA or exercise in a walking intervention. This type of positive coping strategy to decrease pain has been described in previous exercise interventions (Eccleston and Crombez, 1999). However, it was not explored in the reviews using walking to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

Combination treatment

In two reviews that examine walking as a treatment for CLBP, minimal research has been done on walking exercise combined with manual physiotherapy as a treatment (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015). In the reviews, walking has also been combined with education on CLBP or additional exercises (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). Two studies showed pain reductions when walking was combined with manual therapy (Mirovsky et al., 2006; Karadeniz et al., 2014). A RCT investigating the effects of walking combined with vertical ambulatory traction compared with vertical ambulatory traction alone, found that pain had reduced significantly ($p<0.001$) more in the group with walking exercise addition (Mirovsky et al., 2006). A RCT using heart rate monitors to track pacing comparing treadmill walking to over ground walking found pain reductions in both groups (Karadeniz et al., 2014). However, manual therapy factors appear in these walking treatments, where both groups had physiotherapy five times per week and hydrotherapy twice a week for four weeks (Karadeniz et al., 2014). Interactions of factors involved in physiotherapy and hydrotherapy may have positively influenced the pain outcomes. Combination treatment was not trialled against walking or physiotherapy alone. It has not yet been deduced if walking combined with physiotherapy shows statistically or clinically significant differences compared to both physiotherapy and walking independently. A later review which included four studies with CLBP participants used walking combined with other exercises (Sitthipornvorakul et al 2018). This combination of treatments was not studied in the current RCT.

2.7 Summary

Physiotherapy modalities used in UK NICE guidelines and by the majority of South African physiotherapists involve manipulation, massage, and exercise. There is a paucity of evidence supporting walking as an exercise, either used alone or combined with physiotherapy, and for this reason, together with available physiotherapy treatments, it merits further investigation. Consideration of baseline PA and guidance of the time spent walking as an exercise, and the number of steps taken should be reviewed when choosing walking as a CLBP treatment when comparing physiotherapy treatments to it. CLBP may be considered as a heterogeneous entity with pain phenotypes including nociceptive and neuropathic CLBP (Smart et al., 2012a; Baron et al., 2016). For this reason, including pain phenotypes in the modelling process may be a point of departure for future investigation of CLBP cohorts. Following this review of the literature, requirements for studies using walking as a CLBP treatment include:

- Improved objective documentation of activity in all treatment groups
- Improved documentation of walking intervention amount and duration.
- Examining patients' pain expectations relative to outcome
- Examining walking as a sole intervention and as an intervention coupled with usual care physiotherapy
- Explicit definitions of allocated usual care physiotherapy
- Examining physiotherapy treatment content when compared to walking
- Examining the role of supervision as number of visits
- Sample size representing a fully powered RCT
- Specifying subgroups within CLBP (pain phenotypes) as a covariate for modelling purposes

Following this review of the literature, requirements for studies using walking as a CLBP treatment include the use of three treatments defined as: 1) Usual care/ Physiotherapy (P); 2) Pedometer driven Walking intervention (W); and 3) Usual care/ Physiotherapy and Pedometer driven Walking intervention (PW), to explore their effects on pain, disability, kinesiophobia and pain catastrophizing outcomes. The cohort will be examined in the context of nociceptive and neuropathic pain phenotypes in the modelling process.

Chapter 3: Methods

This chapter describes the methods used in the feasibility and main study.

3.1 Ethics

Ethical approval for this randomised controlled trial (RCT) was granted from the University of Bath Ethics Committee on the 10th August 2015, (REACH reference number EP 15/16 18) (Appendix 1). This ensured that 'good clinical practice' was maintained throughout, with refusal or participation in the RCT not compromising clinical care for any of the participants. The RCT was retrospectively registered on the Pan African Clinical Trials Registry (PACTR) on the 5th June 2016 (PACTR201606001660285) (Appendix 2). Patient sensitive information was stored in locked cupboards. Participants were anonymised using numerical reference numbers.

3.2 Intervention planning: Evidence supporting design of treatment groups

Methodological differences in walking interventions to treat CLBP and usual care physiotherapy are noted in previous studies (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). This has made it difficult to assess the efficacy and or effectiveness of such a treatment. To address these disparities, the current RCT has used a randomised controlled design, comparing a pedometer-based walking intervention partly supervised by physiotherapists against usual care (physiotherapy treatment) and a combination of the two. The treatment proposed in this RCT was extracted from (inter)national clinical guidelines, clinical expert opinion, and systematic reviews for treatment of CLBP (National Collaborating Centre for Primary Care [UK], 2009; Liddle, Baxter and Gracey, 2009; Hendrick et al., 2010; Koes et al., 2010; American College of Sports Medicine, 2011; Naidoo et al., 2012; American College of Sports Medicine, 2013). An overview of the proposed three treatment groups is described in Table 20. The treatments in the study were: Usual care (physiotherapy) (**P**); Pedometer-based walking intervention (**W**); and Usual care (physiotherapy) and pedometer-based walking intervention (**PW**)

Table 20: Overview of three treatment groups planned for the main RCT.

	Treatment groups		
Components of treatment	**Usual care /Physiotherapy treatment (P)**	**The pedometer-based walking intervention (W)**	**Combination treatment (PW)**
Usual care/ Physiotherapy treatment	10-15 minutes of lumbar and gluteal massage. 5-10 minutes of Maitland spinal vertebrae mobilisation 10 minutes of Lumbar stabilisation exercise consisting of sub-maximal isometric transversus abdominus contractions. These would be encouraged to be done at home unsupervised daily for 10 minutes daily. CLBP advice/ discussion.	No hands-on treatment	10-15 minutes of lumbar and gluteal massage. 5-10 minutes of Maitland spinal vertebrae mobilisation 10 minutes of Lumbar stabilisation exercise consisting of sub-maximal isometric transversus abdominus contractions. These would be encouraged to be done at home unsupervised daily for 10 minutes daily. CLBP advice/ discussion.
The pedometer-based walking intervention (W)	No hands-on treatment	Standardized daily walking intervention per week. Intervention only supervised when attending a physiotherapy visit, with remaining walks unsupervised. Minimum weekly walking required = 0 days. This was to ensure no increase in sensitization if participants were subjectively too sore. Maximum weekly walking required = 7 days One walk per day (recommended) Weekly Incremental time addition by 10% every week starting with 20 minutes in week 1. Walking intervention discussion CLBP advice/ discussion. Adherence and Incremental walking intervention advised for 12-weeks (unless pain increases)	Standardized daily walking intervention per week. Intervention only supervised when attending a physiotherapy visit, with remaining walks unsupervised. Minimum weekly walking required = 0 days. This was to ensure no increase in sensitization if participants were subjectively too sore. Maximum weekly walking required = 7 days One walk per day (recommended) Weekly Incremental time addition by 10% every week starting with 20 minutes in week 1. Walking intervention discussion CLBP advice/ discussion. Adherence and Incremental walking intervention advised for 12-weeks (unless pain increases)
Treatment number	3 – 9 Treatments with a physiotherapist. A tenth physiotherapy visit was added to obtain incomplete outcome measures or pain and activity diaries not submitted.	3 – 9 Treatments with a physiotherapist. A tenth physiotherapy visit was added to obtain incomplete outcome measures or pain and activity diaries not submitted.	3 – 9 Treatments with a physiotherapist. A tenth physiotherapy visit was added to obtain incomplete outcome measures or pain and activity diaries not submitted.
Treatment duration	12-weeks	12-weeks	12-weeks

NICE guidelines support up to nine physiotherapy treatments of usual care and exercise over a 12-week period for non-specific chronic lower back pain NSCLBP (National Collaborating Centre for Primary Care [UK], 2009). As no guidelines existed in the South African physiotherapy context at the beginning of this RCT, UK NICE guidelines were chosen as the framework to base treatment type and frequency on in this RCT.

All participants were encouraged to visit the physiotherapist during the 12-week RCT, thereby every group was designed to have an opportunity to have a therapeutic relationship between participant and physiotherapist. All three treatment groups had face to face physiotherapist interaction with each participant. All three treatment groups received advice and education. A form of exercise for CLBP treatment was done in all three treatment groups (isometric lumbar stabilization exercise, a pedometer-based walking exercise, or a combination of both exercises). Exercises were also partly supervised. During physiotherapy visits in all three treatment groups, the included exercises were supervised, however participants were advised to continue with the supervised exercise at home daily on advice of the physiotherapist, therefore unsupervised at home.

Advice and education to participants given by physiotherapists in all three treatment groups was based on current knowledge and attitudes towards CLBP, used by the physiotherapists prior to the RCT. During physiotherapy visits, all participants were verbally educated about chronic pain, benefits of the treatment they were receiving for their CLBP, addressing specific participant questions regarding their treatment, and encouraging participants to continue with the three-month course of the RCT. While encouraging participation for 12-weeks, all participants were encouraged to maintain their participation in exercises advised and continue making regular appointments depending on the participant's choice on appointment frequency providing it was between three and nine appointments.

The two interventions of P, and PW were tested against the W intervention. A protocol was developed to explicitly define the three treatment groups used in this RCT (Table 20). If participants received the usual care intervention modalities (P or PW), they would receive spinal manipulation, massage, and isometric lumbar stabilization during every treatment visit (Table 20). These modalities were defined a priori which contrasts with other similar studies (Torstensen et al., 1998; Hurley et al., 2015). The justification for treatment protocol was based on CLBP treatments used, in previous studies (Torstensen et al., 1998; Maher, 2004; Koes et al., 2010; American College of Sports Medicine, 2011; American College of Sports Medicine, 2013). Standardized treatment is demonstrated in Table 20. If participants received a pedometer-based walking intervention, only the defined walking intervention would be used (Table 21).

Table 21: Summary of proposed pedometer-based walking intervention

Week	Duration of daily walking in the walking intervention (minutes)
1	20
2	22*
3	24*
4	26*
5	28*
6	30*
7	32*
8	34*
9	36*
10	38*
11	40*
12	42*

* If pain intensity increased, participants were to attempt doing the amount of walking daily in the previous week.

One exercise intervention in this RCT was a pedometer-based walking exercise intervention. It was performed in isolation (W), and in combination with usual care (PW). It was a planned duration intervention, however measuring step counts and distance for all three treatment groups would provide an objective measure between and within groups for this. All participants were instructed in and requested to complete a daily pain and activity diary (Appendix 3 and 4). Table 22 describes the content of these diaries, by treatment group.

Table 22: Pain and activity diaries (A and B) used in the 3 RCT treatments

Pain and activity diary used	Treatment group	Information from diary
Diary A	Usual care/ Physiotherapy (P)	Pain Intensity VAS Total daily steps Total daily kilometres
Diary B	Pedometer-based walking intervention (W)	Pain Intensity VAS Total daily steps Total daily kilometres Walking intervention steps for the day Walking intervention kilometres for the day Minutes spent doing walking intervention for the day
Diary B	Usual care/ Physiotherapy treatment and pedometer-based walking intervention (PW)	Pain Intensity VAS Total daily steps Total daily kilometres Walking intervention kilometres for the day Minutes spent doing walking intervention for the day

Three physiotherapy visits were allocated to collect outcome measures at baseline, 6-week and 12-week follow-up. Aside from the three mandatory physiotherapy visits to complete outcome measures, the number of physiotherapy visits in this RCT was discretionary. In the context of South African physiotherapy private practice, participants could choose the number of physiotherapy visits they would like. Two primary studies using walking to treat CLBP used the approach allowing the discretion of the treating physiotherapist for when and how often to treat participants/ schedule the number of physiotherapy visits (Eadie et al., 2013; Hurley et al., 2015). The number of physiotherapy visits was not included as an independent variable in the analysis of previous reviews that used a walking intervention for the treatment of CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). However, in the current RCT, it was included as an independent variable. The primary researcher felt the frequency of physiotherapy, which was not previously investigated in studies using walking to treat CLBP, may affect outcomes for pain, disability, kinesiophobia and pain catastrophizing.

Participants in this RCT as in other CLBP walking RCTs using a walking and usual care, were advised to continue normal medication intervention (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015).

Justification to define usual care/ physiotherapy treatment group

Previous systematic reviews of walking to treat CLBP refer to heterogeneous applications of individual physiotherapy modalities as usual care (Hendrick et al., 2010). In this RCT, P treatment group is described in Table 20.

An evidence-based decision was taken to use spinal manipulation, massage, exercise (isometric lumbar stabilization) and advice to treat CLBP in this RCT (National Collaborating Centre for Primary Care [UK], 2009; Liddle, Baxter and Gracey, 2009; Naidoo et al., 2012). These modalities for the treatment of CLBP are used in parallel by physiotherapists implementing UK NICE guidelines and typical South African physiotherapists (National Collaborating Centre for Primary Care [UK], 2009; Liddle, Baxter and Gracey, 2009; Naidoo et al., 2012).

Justification to design a pedometer-based walking intervention treatment group

A Literature search of eleven different countries guidelines for the treatment of CLBP showed them to be generally similar, supporting staying mobile and increased physical activity (Koes et al., 2010). Staying mobile can be measured by step count therefore having walking compared with usual care physiotherapy and to a combination all while measuring the step count between and within all three treatment groups, may explain the levels of PA that occurred when using the treatments in this RCT. Systematic reviews concluded it would be prudent to encourage people to use walking to treat CLBP

Hendrick et al., 2010; Sitthipornvorakul et al 2018). The recommendations in the former review were assessing participant specific characteristics, objectively measuring walking and the review had no evidence-based walking programs used in their methodology. In the current RCT, daily steps counts were monitored and recorded by each participant using a pedometer and recorded in a pain and activity diary (Table 22). Step counts were presented as weekly steps taken. The current RCT also measured patient specific characteristics, phenotyping participants CLBP into nociceptive and neuropathic pain phenotypes, and utilized biopsychosocial outcome measures (kinesiophobia and pain catastrophizing). This would aid understanding into objectively measured step counts and these patient specific characteristics.

The ACSM provides evidenced based guidelines for walking for health, which are internationally recognised. Guidelines for starting a walking programme, published by The American College of Sports Medicine (American College of Sports Medicine, 2011) were used to develop the pedometer-based walking intervention methodology for this RCT (Table 21). Incremental increases in walking time duration were utilized. The methodology devised in this RCT was to increase PA levels in two of the treatment groups (P and PW) using walking as an exercise.

ACSM recommendations state the duration of a walking programme is between 10-30 minutes (American College of Sports Medicine, 2013). At baseline participants in this RCT were classified as insufficiently active. That is, performing less than 150 minutes of exercise per week (Warburton and Bredin, 2017; Fielding et al., 2017; Exercise is Medicine, 2019). By asking participants in the pedometer-based walking interventions to walk for 20 minutes, seven days a week for the first week in the RCT, the total walking exercise time required after one week's participation would still be classified as insufficiently active (totalling 140 minutes per week). As for step counts achieved, according to the literature recommended daily step counts for populations with disabilities or chronic illness are 4000-6000 steps per day (Tudor-Locke and Myers, 2001b).

Participants were advised to walk daily at a moderate intensity providing the intensity did not increase their pain intensity. In line with ACSM recommendations, participants were requested to increase their advised daily walk by two minutes each week (10%), over the 12-week intervention period (Table 21). This walking advise would classify participants as no longer insufficiently active by week two of the pedometer-based walking interventions. They were not excluded from the RCT if pain prevented walking. The intervention was designed to encourage exercise through walking but to not increase peripheral and central sensitization, resulting in pain. Objective measures of number of steps taken per week from baseline to 12-week follow-up would be compared between treatment groups used in the RCT.

Participants were requested to try and maintain compliance with time and frequency of the pedometer-based walking intervention but not to continue with the walking intervention for the day if pain levels increased. An additional instruction to this was to continue with the pedometer-based walking interventions when pain intensity decreased or reached ambient/ previous levels. If, or once decreased pain levels were reached, the participants were advised to begin walking at the daily time duration prior to the increase in pain, unless this was reached in week-one. If so, participants were to start walking again at the 20-minute duration. Regardless of the exercise prescription, participants were not excluded if they walked less than the recommended amount (Table 21). This may have been due to other pragmatic reasons (e.g.: sickness, work or family duties, unforeseen obstructive circumstances such as motor vehicle accidents). To standardize the pedometer-based walking interventions, participants were encouraged not to exceed the amount of walking advised. Adherence to the pedometer-based walking interventions would be recorded over the 12-week RCT. Participants in the W and PW groups would complete diaries with total daily step counts, distance covered, and time taken to complete the daily pedometer-based walking intervention.

All participants in the W treatment group attended physiotherapy visits (Table 20). In previous studies comparing a walking intervention and usual care, participants used telephone contact as physiotherapy visits (Hurley et al., 2015; Eadie et al., 2013; Torsensen et al., 1998). Participants in the W treatment group would not receive telephone calls by the physiotherapist every one to two weeks but attend a face to face physiotherapy visit. Furthermore, every participant in the W and PW treatment groups went for a minimum of one mandatory supervised walk with the physiotherapist during the 12-week RCT to ensure walking was done at a moderate pace. The remainder of the physiotherapy visits could be a supervised walk together with the physiotherapist with advice on their pain, or advice on CLBP, without including a supervised walk. This decision was based on whether the participant had already completed their daily walk or if they felt pain intensity prevented walking on the day. Thus, the pedometer-based walking intervention (W and PW) was only partly supervised. Physiotherapy visits included discussing their progress or problems around any pain and disability, or a face to face meeting and discussion about their progress or problems encountered in the walking intervention. The decision by the patient to not increasing the walking time as per Table 21 was based on increased participant pain and disability since the last treatment preventing desired increased walking time. During physiotherapy visits participants were encouraged to make further physiotherapy appointments based on their perceived need for support.

Justification to design combined usual care and pedometer-based walking intervention group.

This treatment design was an additive treatment combining both the stand-alone pedometer-based walking intervention (W) and stand-alone usual care treatment (P). The participants randomised to

this treatment were asked to complete the pedometer-based walking intervention and attend up to nine physiotherapy visits for massage, manipulation, and isometric transversus abdominus exercise, over the RCT period. The same education and advice on CLBP as used in the former treatment groups was incorporated in the PW treatment. Any problems the participants encountered as in the pedometer-based walking interventions would be addressed within the physiotherapy treatment.

Strategy to ensure participant safety

For ethical reasons and to prevent increased sensitization, instructions were given to physiotherapists not to persevere with any treatment that was causing increased pain.

All the physiotherapists involved in this RCT were trained to instruct the participants to stop their pedometer-based walking interventions if pain increased. The participants were instructed to start the pedometer-based walking interventions again only if pain ceased or returned to pre-treatment levels. Once pain had reached these levels, the pedometer-based walking interventions would continue at the time duration used one week prior to the pain increasing. The incremental 20% time increase for every second week would be applied for the remainder of the intervention.

Physiotherapists were instructed not to cause participants increased pain when using massage, manipulation, and isometric transversus abdominus exercises. Adverse events which were experienced by participants were recorded in the pain and activity diary. Participants were free to withdraw from the RCT at any time if they were unable to cope with exercise for any physical reason, or if pain became severe.

3.3 Measures

Initial measures taken were only completed at baseline. Outcome measures for the trial were completed at baseline, at six-weeks and at 12-weeks. Visual Analogue Scale Pain and Activity diaries were completed daily by participants.

Baseline questionnaires, screening tools and lumbar assessments

Measures recorded at baseline only are described in Table 23.

Table 23: Baseline collection of demographic, clinical, and anthropometric measures used in the RCT

	Description	Time stage at which measure was obtained	Reference	Method of measurement:
Health history & demographic questionnaire	Health History demographic questionnaire used to gather demographic data	Baseline	Designed by the primary researcher	Questionnaire: Self-measurement by participant Anthropometric data height, body mass and stride length were recorded by the physiotherapist.
Pain phenotype screening tool	painDETECT questionnaire 0-12 unlikely to have a neuropathic component 13-19 Ambiguous but a neuropathic component is likely >19 a neuropathic component is likely	Baseline	Freynhagen et al., 2006	Self-measurement by participant
Patient centred outcome questionnaire NRS	Questionnaire measuring participant expectation of pain intensity at follow-up measured on a NRS (0-100) 0=no pain; 100=worst imaginable pain	Baseline	Sanderson et al., 2012	Self-measurement by participant
Lumbar assessment	To record professional examination of participant and exclude any red flags (infection, fracture, cancer). Postural and movement observation; history of participant symptoms; basic physiotherapist neurological tests; joint and muscle palpation; and special tests to confirm CLBP	Baseline	Maitland, 2001	Observational assessment by attending physiotherapist at baseline and written into lumbar assessment form

Health history and demographic questionnaire

This questionnaire was designed to assess participant eligibility for the RCT (Appendix 5). In addition, demographic data were recorded by participants at baseline in this questionnaire. Furthermore, participant anthropometric height, body mass and stride length were recorded by the physiotherapist. Stride length, measured with a standard tape measure in centimetres, was assessed as a mean of ten strides. Digital home bathroom scales were deemed fit to provide accurate and consistent body mass measurement (Yorkin et al., 2013). Body mass was measured in kilograms using new digital Beurer scales with participants fully clothed but without shoes on. Height was recorded in centimetres using a fixed vertical tape measure mounted on the wall.

PainDETECT questionnaire

Prior to a lumbar evaluation and outcome measure assessments, categorizing participants by their dominant pain phenotype was conducted using the painDETECT questionnaire. Pain phenotyping would be included in the modelling process. This is a validated instrument which enables a physiotherapist to identify nociceptive and neuropathic pain phenotypes in patients with CLBP (Freynhagen et al., 2006), it is further described in appendix 6. The painDETECT was developed and validated in German and is available in English (Freynhagen et al., 2006). The tool has a sensitivity of 85% and specificity of 80% for correctly classifying CLBP. It has good internal consistency for the detection of dominance of neuropathic pain phenotype; with a Cronbach's alpha > 0.83 and excellent test-retest reliability, showing an ICC = 0.93 (Mathieson and Lin, 2013).

The painDETECT consists of a nine item self-report questionnaire. Pain patterns are described in the questionnaire by seven weighted sensory descriptor items (0 = 'never' to 5 = 'very strongly') and two items relating to the spatial (radiating) and temporal characteristics. The score ranges from 0-38. The principal researcher used the score interpretation to phenotype participants according to their pain. Participants LBP scoring 0-12 were classified as having nociceptive CLBP. Participants having scores of 13-38 were classed as having a neuropathic pain component, or neuropathic CLBP (Freynhagen et al., 2006). The questionnaire indicates that scores ≤12 are unlikely to have a neuropathic component (<15%), whereas a score of ≥19 suggests that pain is likely to comprise of a neuropathic component (>90%). The score from 13 - 18 is categorized as ambiguous in the questionnaire and that a neuropathic component may be present (Freynhagen et al., 2006). A cut-off point at 13 was decided upon by the principal researcher in the current RCT. To summarise, scores from 0-12 were classified as nociceptive CLBP; and scores 13-38 as neuropathic CLBP in this RCT.

Participant expectation of pain reduction at follow-up

At baseline, each patient's expectation of their pain at 12-week follow-up, was assessed using the Patient Centred Outcome Questionnaire seen in appendix 7 (PCOQ). The PCOQ has five sections where initially participants indicate their usual levels (during the past week) of pain, fatigue, emotional distress, and interference with daily activities on 101-point NRS (0 = none, 100 = worst imaginable) (Sanderson et al., 2012). Only the expectation of pain intensity scores gathered at baseline were analysed in this RCT. In pilot data of 21 spinal pain patients, there was acceptable test-retest reliability with reliability values ranging from 0.84 to 0.90 ($p<0.001$) (Brown, 2004). The PCOQ has good concurrent validity with standardized measures of pain, mood, and disability (Brown, 2004).

3.4 Lumbar assessment

There is no current gold standard in lumbar assessment. The lumbar assessment used followed a Maitland style approach (Maitland, 2001). The lumbar assessment at baseline included postural and movement observation; taking a detailed history of patient symptoms; basic physiotherapist neurological tests; joint and muscle palpation; and special tests to confirm that the diagnosis of CLBP was correct. The assessment is further described in Appendix 8.

Outcome Measures

Table 24 describes the outcome measures selected for this RCT. Outcome measures were selected to evaluate biopsychosocial aspects and functioning of participants. The outcome measures were completed by participants at baseline and at six and 12-week follow-up (Table 24).

Table 24: Outcome measures taken at baseline, six-weeks and 12-weeks follow-up in the RCT.

	Definition	Description of outcome measure	Reference	Time stage at which outcome measure was administered	Method of measurement:
Outcome measures	Primary outcome measure	Numerical rating scale of pain intensity (NRS) (0-100) 0=no pain; 100=worst imaginable	Ostelo and de Vet, 2005; van der Roer et al., 2006	Baseline; Six-weeks follow-up; 12-weeks follow-up	Self-measurement by participant
	Secondary Outcome measures	Oswestry disability index (ODI). A greater score indicates a greater level of disability.	Bombardier, Hayden and Beaton, 2001; Fairbank and Pynsent, 2000		
		Tampa Scale of Kinesiophobia (TSK). A greater score indicates a greater level of kinesiophobia.	Vlaeyen et al., 1995		
		Pain Catastrophizing scale (PCS). A greater score indicates a greater level of catastrophic thinking.	Sullivan, Bishop and Pivik, 1995		

Numerical rating Scale of Pain intensity

Pain intensity is typically measured by specific questionnaires, which are used in physiotherapy and CLBP studies (Heymans et al., 2006; Kääpä et al., 2006). Reliability and validity of the Numerical rating scale (NRS) and Visual Analogue Scale (VAS) are well established to assess pain intensity (Haefeli and Elfering, 2006; Mannion et al., 2007; Boonstra et al, 2016; Suzuki et al., 2020). The minimum clinically important difference (MCID) is an indicator of clinical effectiveness. A 20% change is recognized as a MCID on the NRS (Farrar et al., 2001; Haefeli and Elfering, 2006).

A review by Mannion et al., (2007) summarizes a NRS to be the most practical index when measuring pain intensity in LBP due to responsiveness to change, ease of administration and sensitivity (defined by the number of available response categories). Thus a NRS was used to measure the primary outcome of pain intensity (Appendices 9, 10, and 11). Some studies have a minimum level of pain as inclusion criteria. However, the RCT using walking to treat CLBP advocated no minimum due to the recurrent nature of CLBP having episodes of little, or no noticeable LBP (Hurley et al., 2015). Anchoring statements for the outcome for pain intensity and time frame measuring pain must be well defined (Ogon et al., 1996). The NRS used in this RCT was anchored with the statement of your usual pain being none to worst imaginable on a scale from 0-100 (0 = no pain and 100 = worst imaginable).

The Oswestry Disability Index

The Oswestry Disability Index (ODI) is used to measure a person's disability, relating from spinal disorders (Fairbank and Pynsent, 2000). It is considered the best index for measuring how lower back or leg pain affects an individual's ability to manage in everyday life (Fairbank and Pynsent, 2000; Bombardier, Hayden and Beaton, 2001). Often patients report continued disability despite varying pain intensities or the absence of pain (Rantanen, 2001). The ODI has been used in LBP studies, in South African populations (Kruger and Bilson, 2014). The ODI was widely used to measure CLBP disability in the two systematic reviews using physiotherapy or walking as a treatment (Hendrick et al., 2010; Lawford, Walters and Ferrar., 2015). A large cohort study of 837 Finnish outpatients with LBP aimed to investigate the psychometric properties of the ODI (Saltychev et al., 2017). Data from the study showed the ODI to be an internally consistent (α=0.85), unidimensional scale (measuring functional level and nothing else) with overall excellent construct validity and ability to discriminate between disability severities (Saltychev et al., 2017). Supporting its construct validity, it has been used to validate the Pain Disability Index, The Low Back Outcome Score, has moderate correlation with pain measures (NRS); and is a better predictor of return to work than mechanical methods of lumbar spine assessment (Roland and Fairbank, 2000).

The ODI comprises of 10 sections each covering one of the following domains mentioned in Table 25. Within each section there are six statements ranked zero to five. The anchors are domain specific although zero score indicates no disability associated with a domain statement (e.g.: lifting, walking). A score of five indicates pain prevents a participant from executing that domain. The participant chooses which of the six statements within each section that best applies to them. A score is generated as a percentage. The higher the score (%), meant a greater perceived disability. Table 25 describes the associated level of disability, by score for the total included domains of disability. The MCID for the ODI is 10% (Ostelo et al., 2008). Details of the items included, and the formula used to calculate the score are described in appendix 12.

Table 25: ODI levels of disability relative to contributing domains.

Domains of the ODI	Total ODI score (%)	Level of disability
1. Pain intensity 2. Personal care (such as washing, dressing) 3. Lifting 4. Walking 5. Sitting 6. Standing 7. Sleeping 8. Sex life (if applicable) 9. Social life 10. Travelling	0-20	Minimal disability
	21-40	Moderate disability
	41-60	Severe disability
	61-80	Crippled
	81-100	Bed bound or magnifier

(Roland and Fairbank, 2000)

The Tampa Scale for Kinesiophobia (TSK)

The Tampa Scale for Kinesiophobia (TSK) measures fear of movement/ (re)injury and is used in studies supporting the fear avoidance model (Vlaeyen et al., 1995). The fear avoidance model describes CLBP patients who acquire a fear of movement after an initial acute phase of LBP. After suffering an initial injury, one may become fearful of movements that hurt and hence avoid further activity. This in turn may result in CLBP from acute LBP (Crombez et al., 2012). The TSK is used to assess fear and avoidance beliefs in movement and re-injury studies, specifically in CLBP (Kori, Miller and Todd, 1990; Vlaeyen et al., 1995). The TSK scale has been well validated in chronic pain populations including musculoskeletal pain, neuropathic pain, headache, and abdominal pain (Vlaeyen et al., 2002; Tkachuk and Harris., 2012). In CLBP studies, construct validity of the TSK was supported by Roelofs et al., (2011). The reliability of the TSK was examined in a review of 12 articles, with four English variations of the TSK (Lundberg et al., 2011). Various English language versions contain four, 11, 13, or 17 items. The reliability in four English articles ranged from acceptable (α=0.71) in the four-item version subscale TSK to high (α=0.86) in the 13-item version (Lundberg et al., 2011). The English 17 item version reliability was not reported in Lundberg et al., (2011).

The TSK used in this RCT used 17 statement questions (Woby et al., 2005; Woby et al., 2008.) The questionnaire asks individuals to rate the extent to which they agree with the statements. Each of the 17 questions have four points ranging from 1 = strongly disagree to 4 = strongly disagree. Four questions are reverse scored (4; 8; 12; 16). Total scores range from 17 – 68. A high value on the TSK indicates a high degree of kinesiophobia. A cut-off score of 37 or greater is considered as clinically relevant (Vlaeyen et al., 1995). Since the current RCT was underpinned by an exercise intervention involving movement (walking), evaluating changes in kinesiophobia using the TSK (Appendix 13) was deemed appropriate.

Pain catastrophizing scale (PCS)

Pain catastrophizing is defined as an exaggerated, negative mental state that can occur in patients in response to an actual or anticipated painful experience (Sullivan et al., 2001a). The Pain Catastrophizing Scale (PCS) is made up of three factors: rumination, helplessness and magnification of painful events. The contribution of catastrophizing in physiotherapy studies to CLBP is well established (Peters, Vlaeyen and Weber, 2005; George, Valencia and Benecluk, 2010). Measuring pain catastrophizing in CLBP patients is important with self-management strategies, as proposed by a walking intervention (Nicholas et al., 2012). With various models implicating pain catastrophizing in CLBP, the PCS was chosen as a relevant outcome measure of catastrophic thinking in relation to chronic pain in this RCT (Appendix 14).

Three studies conducted on pain outpatient samples found strong evidence of validity for the PCS (Osman et al., 1997). Demonstrating internal consistency, in a study of 425 undergraduate Canadian psychology volunteers, the Cronbach's alpha values reported for the total PCS (α= 0.87) was very good (Sullivan, Bishop and Pivik, 1995). Aiming to evaluate the assumption that the PCS taps a single construct characterized by three dimensions, a study examined evidence for concurrent and discriminant validity of the PCS in an American community sample (n=215) (Osman et al., 2000). High and significant inter-correlations were found between factors of rumination, helplessness and magnification which make up pain catastrophizing. Regarding construct validity, this appeared to be the first finding in the literature confirming the assumption that the PCS taps a single construct (catastrophizing) characterized by three related dimensions (Osman et al., 2000). The PCS is recommended as a valid and reliable tool in South African pain studies (Morris et al., 2012).

The 13-item questionnaire accurately evaluates pain related magnification, helplessness and rumination (Jensen, Turner and Romano, 2001; Sullivan et al., 2001b; Picavet, Vlaeyen and Schouten, 2002; Lamé et al., 2008; Sullivan et al., 2009; Suzuki et al., 2020). The PCS consists of a five-point scale of 13 items asking the participant to reflect on past painful experiences and indicate the degree to which they have experienced certain feelings or thoughts upon experiencing pain. The points range from a score of zero to four, where zero represents 'no worry at all' and four represents 'worrying all the time' or increased levels of catastrophizing (Sullivan, Bishop and Pivik, 1995). A PCS total score ranges from 0 – 52. A total score of 30 or greater is a cut-off score and represents a clinically relevant level of catastrophizing (Sullivan, Bishop and Pivik, 1995).

3.5 Visual Analogue Scale Pain and Activity diaries

Additional tools to assess pain, daily and step counts were used and are described below.

Pain and activity diaries

Daily pain and activity diaries were used by participants in this RCT (appendix 3 and 4 respectively). Diaries were designed by the principal researcher. These were used to keep record of average pain intensity on a Visual Analogue Scale (VAS), and daily steps taken/ kilometres (completed on a daily basis). Each diary had a page per day for pain intensity and steps/ kilometres (Table 26).

Table 26: Visual Analogue Scale Pain and Activity Diaries

	Screening tool description	Time stage at which measure was obtained	Reference	Method of measurement:
Paper pain and activity Diary	Measuring step count and distance Measuring time taken to walk in walking intervention (W and PW)	Daily (at bedtime)	Diary Designed by the primary researcher	Self-measurement by participant
	Measuring daily average pain intensity. Visual analogue scale (VAS) on a horizontal 100mm line. 0mm=no pain and 100mm=worst pain imaginable	Daily (at bedtime)	Hägg, Fritzell and Nordwall, 2003; Ostelo and de Wet 2005 Diary Designed by the primary researcher	Self-measurement by participant

All participants were provided with and instructed in completing the daily pain and activity diary (baseline to 12-week follow-up). Participants randomised to the W or PW treatment groups were shown how to use pain and activity diary A (Appendix 3). Participants randomised to the P treatment group were shown how to use pain and activity diary B (Appendix 4). The difference between the diaries was that the Walking Intervention component was included in diary A. This design was used to maximise information gathered and to control for walking (as a physical activity) done outside of the walking intervention (exercise). Both diaries were designed for participants to record daily number of steps taken, distance covered, and time taken to complete a walking intervention if randomised to one.

Both diaries included a VAS. Decreasing the need for interviews, patient diaries are an alternative to capturing patient perceptions of their pain (Miller, Pinnington and Stanley, 1999). Thus, in this RCT participants were asked to complete diaries at bedtime in the evening. The diary satisfies part of the biopsychosocial paradigm where the opportunity of the participants experiences to be recorded is on hand (Gatchel et al., 2007).

Visual Analogue Scale (VAS)

To improve supervision, participants were requested to log their daily average pain using the VAS in a pain and activity diary (appendix 3 and 4). The daily VAS was used to record average daily pain from baseline to follow-up. Attending physiotherapists could examine this diary when discussing participant progress on subsequent physiotherapy visits during the RCT. This diary would inform the physiotherapist if pain had increased substantially, hindering the participant's continuation in the RCT. The intention was to concurrently monitor daily average pain intensity in the diary in order to avoid further added walking in the intervention in case this was making the pain worse. This was supported by a review for LBP patients with leg pain, recommending exercising regularly providing this does not increase leg pain (Pedersen and Saltin, 2015). There is no consensus on when and how often to measure pain due to its unreliable duration (Mannion et al., 1999). Physiotherapists could monitor the participant's pain diaries retrospectively since a daily record in the diary would log both pain intensity and frequency. These are often highly correlated (Chang et al., 2003). However, measuring symptom frequency within 24 hours is not supported (Deyo et al., 1992). This supported the decision to log the daily average pain intensity in the diary, and not hourly, as this would be a cumbersome measure.

Using a three 100mm horizontal VAS scales per daily diary page, participant's pain was measured for right leg, left leg and lower back pain. The VAS can be used vertically although the horizontal scale is preferred in CLBP (Ogon et al., 1996). Each VAS being identical, the VAS in this RCT was anchored with a statement of: your average pain being no pain (0mm) - very severe pain (100mm) on a 0-100mm horizontal line (Appendices 3 and 4). A MCID on a VAS in CLBP is around 20mm on a 100mm line, or 20% (Hägg, Fritzell and Nordwall, 2003; Ostelo and de Wet, 2005). Both paper diaries and electronic format VAS compare well (Jamison and Edwards, 2012). However, the purpose of this RCT was to make it generalizable and equitable and so paper diaries were used. The VAS is a validated outcome measure for a CLBP population in South Africa (Kruger and Bilson, 2012)

Objective measure of walking

Pedometers were used to objectively measure walking in each of the intervention groups. These data of steps taken, distance covered, and time taken to complete walking interventions if randomised to one were recorded by participants in pain and activity diaries (Appendix 3 and 4). By using diaries, differences in steps and distance covered could be identified in the walking done by treatment groups involved in walking intervention (W and PW) and those not using a walking intervention (P).

All participants were required to record total daily steps and distance walked, daily, in their activity diaries. Walking interventions (W and PW) used diary A (Appendix 3). Usual care with no walking

intervention used diary B (Appendix 4). The difference between diaries lay in that diary A could be used to log daily time involved in the walking intervention (Table 21).

3.6 Pedometers

In-line with previous studies Omron Walking Style One 2.1 HJ-321-E pedometers were used in this RCT (Tudor-Locke et al., 2011; Rodriguez-Sanchez et al., 2014). These battery-operated pedometers were used to measure daily step count and distance walked (Omron Healthcare INC., 2015).

Fifty OMRON Walking Style One 2.1 HJ 321 E Pedometers were purchased and 30 were supplied as a sponsorship from Omron Europe. The sponsored pedometers were obtained six-weeks after the RCT began.

To accurately measure step count and distance, at baseline each pedometer was programmed by the assigned physiotherapist with the participant's relevant information (height and step length). Participants were instructed in how to change batteries and re-enter their anthropometric data and stride length. The participant was issued with and instructed in the use of the pedometer in conjunction with the Pain & Activity Diary (Appendix 3 and 4). Participants were requested to wear their pedometers from when they got up in the morning until when they went to bed. At baseline, participants were asked to trial the pedometer for the remainder of the day. If any problems with pedometer arose, they were to contact their assigned physiotherapist who would attend to related problems related to the pedometer or diary use. Data from the pedometer were to be entered into corresponding diaries the day after the baseline interviews.

3.7 Training for delivery of interventions of RCT physiotherapists

A month prior to the main RCT, training for the RCT physiotherapists regarding RCT protocols was run for one week. Training was based on the intervention planning by the principal researcher. Training of physiotherapists was to ensure that treatments in Table 20 were rehearsed in order to remain homogenous within treatment groups when delivered by the physiotherapists. RCT physiotherapists underwent one week of training to gain a better understanding of CLBP including treatments available and pain phenotyping. The training involved intervention delivery. This involved rehearsal with the protocol which would be followed from the first physiotherapy visit baseline interview including all questionnaires, pedometer use, and the ability to execute homogenous treatments within each treatment group involved in the main RCT until 12-week follow-up.

The training for RCT physiotherapists incorporated 2 phases:

1. A teaching platform for the physiotherapists involved in delivering the intervention in order to educate them about explaining CLBP, education of potential evidence-based benefits for treatments used in this RCT, and pain phenotyping.
2. A teaching platform to attempt standardizing obtaining baseline questionnaires, involvement in the randomization process through painDETECT completion, performing a standardized lumbar assessment, assisting in completion of outcome measures, delivery of components involved in each treatment group.

Phase 1:

The training was based on the literature review, including UK NICE guidelines on CLBP physiotherapist treatment (National Collaborating Centre for Primary Care [UK], 2009). Understanding pain phenotyping was included (Freynhagen et al., 2006).

Phase 2:

Training was provided on:

1. Baseline screening of inclusion criteria, explanation of the RCT, signing of the consent form, completing health history questionnaires, and completing the painDETECT phenotyping questionnaire.
2. Completing a lumbar spine assessment.
3. Liaising with the practice secretary to obtain treatment randomization according to the painDETECT score.
4. Assisting participants complete outcome measures at baseline and follow-up outcome measures at six-weeks, and at 12-week follow-up.
5. All three treatments and included practice of improvised CLBP education, massage, manipulation, isometric lumbar stabilization exercise and supervised pedometer-based walking as to be used in the RCT.
6. Completing pain and activity diaries were rehearsed whilst trialling the pedometer in order to teach RCT participants how to use this effectively.

3.8 Feasibility study of procedures involved in the main randomised controlled trial

Once all training had been delivered, a feasibility study of the procedures to be used was conducted. This was run three weeks prior to the main RCT. Results from the feasibility study run would be used to streamline RCT processes used in the main RCT if necessary.

The feasibility study consisted of running through procedures that would be used in the main RCT in the three treatment groups (P, PW and W). The principal researcher invited five physiotherapists and six healthy lay members of the public to participate in the one week-long feasibility study. The principal researcher would also participate in this feasibility study. All 11 participants were interviewed by the principal researcher to illuminate the purpose of the feasibility study and would gain their consent to participate. The exact RCT procedure including informed consent, questionnaires, pedometers and treatment groups all as described previously were included in the feasibility study.

The physiotherapists would fill two roles; firstly as a treating physiotherapist role, followed by a role as a participant treated by a fellow physiotherapist. The role as a treating physiotherapist was done by each physiotherapist in order to experience treating the P, PW and W procedures over a one-week period. During this week each physiotherapist would treat either another physiotherapist or lay member of public designated by the principal researcher to one treatment group per individual participant until the treating physiotherapist had experienced treating one allocated participant three times in a week for each individual treatment group.

In some instances, participating members of the lay public may have had to experience two or three different treatment types for every physiotherapist to have trialled the treatments and protocols to be used. Participants may have experienced several different treatments (P, W and or PW) during one day for all treating physiotherapists to complete the experience of treating according to RCT protocol.

The role a physiotherapist played acting as a participant would be to experience the usefulness of the questionnaires, pain and activity diary and pedometer used as well as having received treatment intended to be homogenous for each treatment group (P, W and PW). Each physiotherapist would have to experience being treated by one other physiotherapist three times per treatment group. During the week, the treating physiotherapist would repeat treatment accordingly to W, P, and PW protocol to participants allocated to those treatment groups. The participants were not randomised to treatment groups but allocated ad hoc by the principal researcher. For example:

Physiotherapist treats participant three times as per W treatment group.

Physiotherapist treats participant three times as per P treatment group.

Physiotherapist treats participant three times as per PW treatment group.

And:

Physiotherapist receives treatment as per W treatment group.

Physiotherapist receives treatment as per P treatment group.

Physiotherapist receives treatment as per PW treatment group.

The process involved in the feasibility study and physiotherapist protocol training are described in Table 27.

Table 27: Physiotherapist and participant roles in protocol training during feasibility study.

A	B	C
People involved in the feasibility study	**Steps taken by people involved in the feasibility study**	**Aim of feasibility study**
Physiotherapist role as a treating physiotherapist	1. Treating physiotherapist would explain the purpose of the RCT and answer any questions related to the RCT 2. After the consent was signed, the physiotherapist to assist participant with filling in baseline questionnaire health history and painDETECT. 3. Physiotherapist to complete a lumbar assessment of the participant. 4. Physiotherapist to assist participant with completing baseline outcome measures and pain expectation questionnaire 5. Physiotherapist to teach the participant how to use the daily pain and activity diary 6. Physiotherapist to teach the participant how to use the pedometer 7. Physiotherapist to treat the participant in the procedure of P, PW or W for all three treatments to be completed over a week by each physiotherapist. Three treatments per participant i.e.: resembling baseline, midway, follow-up. 8. Assist participant in completing the outcome measures mid-week (which would be used at six-weeks in the main RCT) and follow-up at the end of the week (which would be used at 12-weeks in the main RCT). These outcome measures were both identical	Each RCT physiotherapist would complete one week of each individual treatment for one participant per treatment. For example: • Treat one participant three times a week using procedure P. • Treat one participant three times a week using procedure PW • Treat one participant three times a week using procedure W Each RCT physiotherapist would treat two members of lay public, and one other physiotherapist involved in the RCT for P, PW or W procedure three times a week. In this way all three treatments would have been practiced three times over a week.

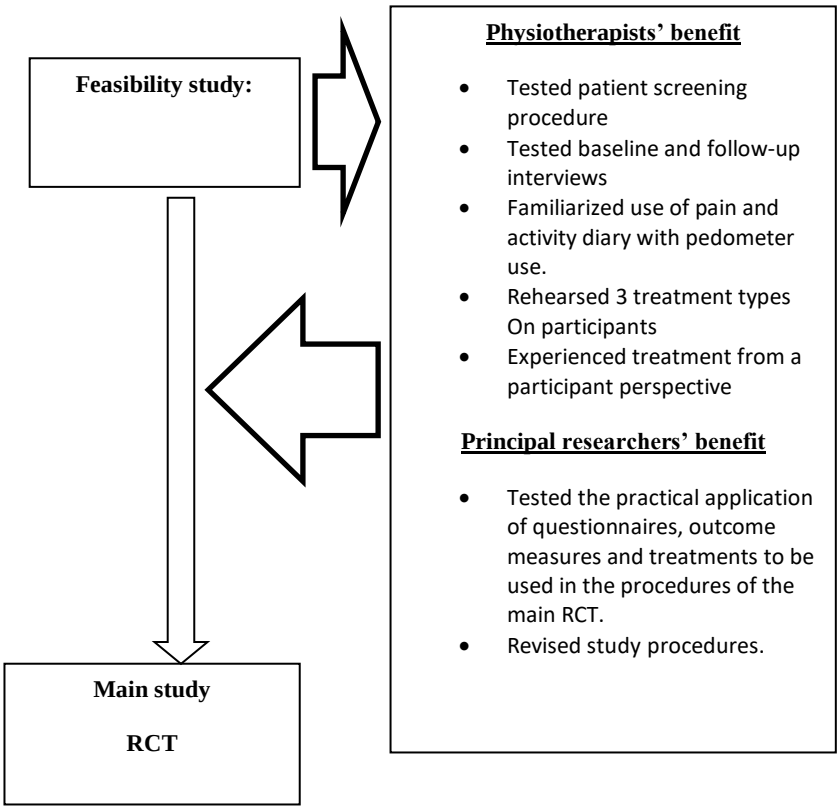
A	B	C
People involved in the feasibility study	**Steps taken by people involved in the feasibility study**	**Aim of feasibility study**
Physiotherapist role acting as a participant.	1. Ask the physiotherapist questions if they felt any important information had been excluded and sign consent. 2. Complete the attending baseline health history questionnaires, painDETECT 3. Undergo a lumbar assessment as a participant by a RCT physiotherapist. 4. Complete baseline outcome measures questions and pain expectation questions 5. Learn how to use a pedometer and the pain and activity diary 6. Participate daily for 1 week in the P, PW or W treatment (i.e.: complete pain and activity diary and receive treatment on allocated days) 7. Complete the outcome measures mid-week (which would be used at six-weeks in the main RCT) and follow-up at the end of the week (which would be used at 12-weeks in the main RCT). These outcome measures were both identical	Each RCT physiotherapist would act as a participant in one of the other RCT physiotherapist's treatments. The physiotherapist acting as a participant would experience either steps 1-5 (column B) as a participant receiving P, PW and W treatments from three individual physiotherapists.
Participant's (lay member of the public) role in the feasibility study	1. After listening to the RCT explanation by an attending physiotherapist ask the physiotherapist questions if they felt any important information had been excluded. Sign a consent form. 2. Complete the attending baseline health history questionnaires, painDETECT 3. Undergo a lumbar assessment as a participant by a RCT physiotherapist. 4. Complete baseline outcome measures questions and pain expectation questions 5. Learn how to use a pedometer and the pain and activity diary 6. Participate daily for 1 week in the P, PW or W treatment (i.e.: complete pain and activity diary and receive treatment on allocated days) 7. Complete the outcome measures mid-week (which would be used at six-weeks six in the main RCT) and follow-up at the end of the week (which would be used at 12-weeks in the main RCT). These outcome measures were both identical	Fulfil the role of the participant with physiotherapy visits per treatment group in one week. The three physiotherapy visits that the participant would complete was arranged by the treating physiotherapist. The number of times a participant could participate was determined in order for each physiotherapist to have assessed and treated one person (either physiotherapist acting as a participant or member of the lay public) for one week, in each procedure (P, PW, W).

After one week of treatment, the participants and physiotherapists were asked to return their completed pain and activity diaries. Baseline and follow-up outcome measures were collected at the first and last appointment respectively. The RCT outcomes were collected by the physiotherapist in the following order:

- Baseline outcome measures: NRS, ODI, TSK, PCS (Appendix 9, 12, 13, 14 respectively) as well as pain expectation questionnaire (Appendix 7) (mimicking baseline in the main RCT)
- Midway outcome measures: NRS, ODI, TSK, PCS (Appendix 9, 12, 13, 14 respectively) (mimicking six-week follow-up in the main RCT)
- Follow-up outcomes measures: NRS, ODI, TSK, PCS (Appendix 9, 12, 13, 14 respectively) (mimicking 12-week follow-up in the main RCT)

All the forms were inspected by the principal researcher in order to see if there was a need to change the procedure. Benefits of the feasibility study are shown in figure 7.

Figure 7: Flow diagram of benefits of feasibility study and physiotherapist training



No changes were made following this process. Key disadvantages of the feasibility study were three-fold:

1. Template for data entry: A template for data entry had not been created for results of the feasibility study
2. Data Analysis: No data analysis had been planned for results gained from the feasibility study
3. No dummy table of results was developed for the feasibility study

3.9 The randomised controlled trial: Main study

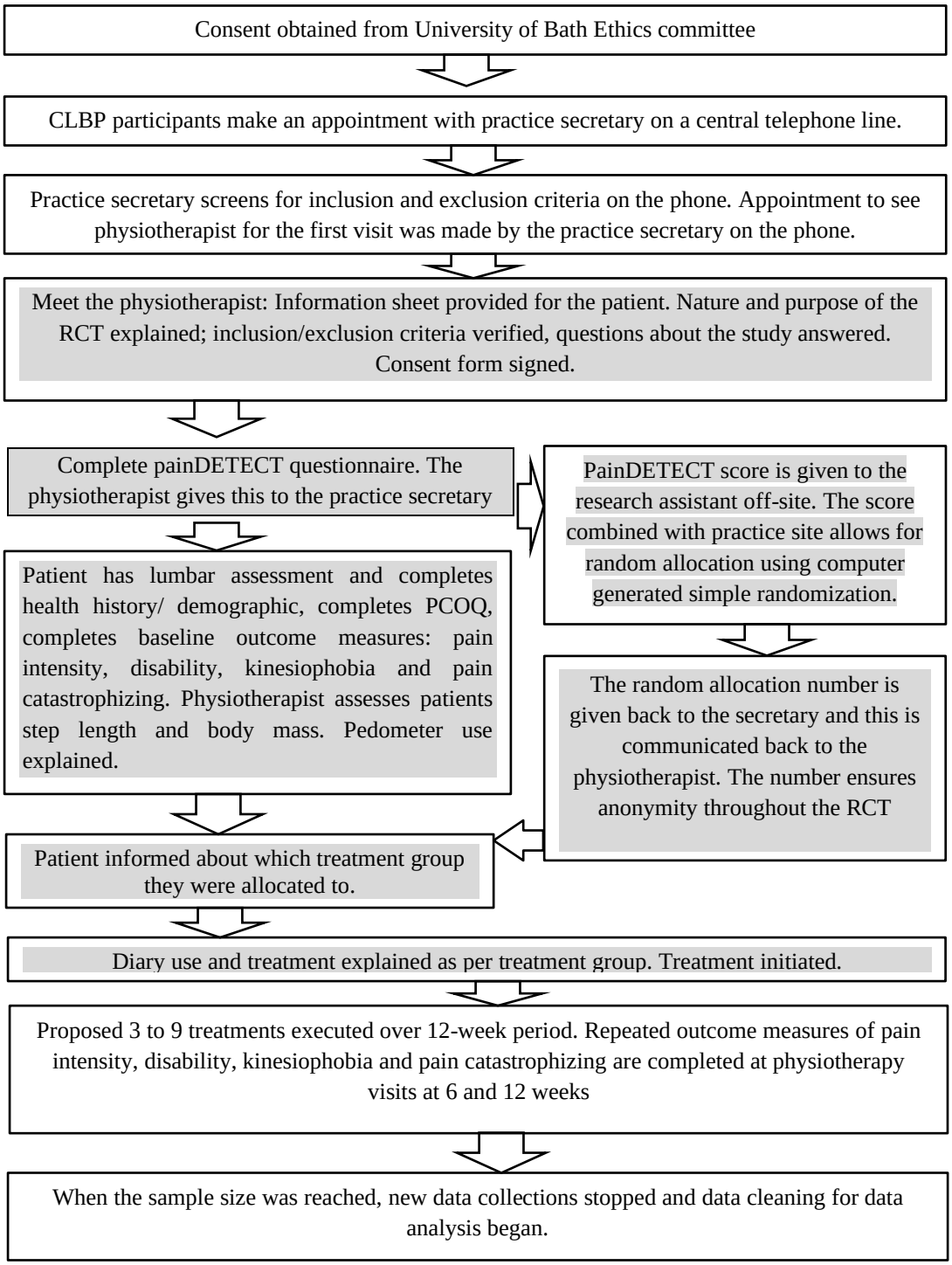
This RCT compared three treatments: P, W and PW.

Primary outcome: A change in baseline global pain intensity using a numerical rating scale between baseline and 12-week follow-up.

Secondary outcomes: A change in baseline disability, kinesiophobia and pain catastrophizing between baseline and 12-week follow-up.

A flow chart detailing the procedures is described in figure 8.

Figure 8: Flow diagram of main study (RCT)



Legend □: Shaded areas in figure 8 explain the first physiotherapy visit. A detailed account of the process from when the participant arrived at the first physiotherapy visit until the completion of the RCT at follow-up is noted in the following procedure chapter.

Participant recruitment, setting, and procedures for the main randomised controlled trial

The following section will detail participant recruitment, setting and recruitment procedures.

Participants included in the RCT recruitment

The inclusion and exclusion criteria are described in Table 28 were adapted from previous studies using walking to treat CLBP used in a systematic review at the inception of the RCT (Hendrick et al., 2010).

Table 28: Inclusion and exclusion criteria

Inclusion & exclusion criteria	Participants
Inclusion criteria	• LBP lasting for longer than 3 months and/or recurring episodes greater than 3 per year. • Less than 150 minutes of intentional exercise per week. • LBP with or without radiation into leg/s. • Participants attending private practice physiotherapy. • Age: 18-65 years. • Participant able to walk a minimum of 20 minutes. • Participant willing to participate in a 12-week RCT, which required a minimum of three and a maximum of nine physiotherapy treatments.
Exclusion criteria	• Current pregnancy. • Knowledge of a malignancy. • Knowledge of rheumatic disease systemically causing lower back pain. • Currently diagnosed with fibromyalgia. • History of serious psychological or psychiatric illness (mild depression eligible for inclusion). • Involvement in a Workman's compensation claim or on-going litigation due to CLBP. • Medically unfit to participate in exercise intervention. • Acute/ recent spinal fractures. • Any other current musculoskeletal injury or contraindication to increasing walking times, including cardiorespiratory or other medical condition limiting exercise tolerance. • Evidence of nerve root, spinal cord, or cauda equina compression indicating signs of neurogenic claudication.

Settings

This was a multi-site RCT. Participants were recruited from the following private physiotherapy practices in Johannesburg South Africa:

1. North-riding Private Practice Neurosurgical Rooms.
2. Randburg Medicross Clinic.
3. Doornfontein Towers West Medical and Dental centre.

Consent for use of these premises was obtained by the researcher before applying for ethics.

An advert was designed by the principal researcher and deemed acceptable by the University of Bath Ethics Committee. It was placed in the waiting areas of all three settings. The advert stated a telephone call should be made on the central booking line with the practice secretary for any enquires. If a potential participant contacted the practice secretary, she would explain the RCT in brief as well as clearly describe the inclusion and exclusion criteria. In line with standard private practice routine, the practice secretary would on an ad hoc basis arrange an appointment at a mutually convenient time for the participant and one of six physiotherapists. The setting that the participant would be seen at was also based on the participants' convenience.

Procedures

Following the plan to design homogenous treatments within each treatment group to be used in this RCT, and having completed a feasibility study of procedures, the main study commenced. The use of measurements and treatments trialled in the feasibility study were deemed appropriate to be used in the main RCT. The procedures involved a team of people invited by the primary researcher, each had specific functions described in Table 29.

Table 29: Research team involved in the main study and their function

Team member	Role
Practice secretary	To take telephone bookings for the RCT, screen the potential participants for eligibility on the phone, and arrange a baseline/ first physiotherapy visit. To communicate the painDETECT score from the baseline/ first visit to an off-site research assistant in order to obtain a random allocation number and treatment which was to be communicated to the physiotherapist. All done during the first visit. To take bookings for additional appointments over the telephone from RCT participants.
Off-site research assistant	To obtain a painDETECT score from the practice secretary and check the random allocation sequence. The off-site research assistant used the painDETECT score and which practice the participant was seen at to find the randomization number and treatment type. The off-site research assistant had access to the random allocation sequence list (Appendix 17) and was blinded to the three treatments. Eligible potential participants were randomised using simple randomization from a previously generated computer sequence. Participants were then assigned with equal probability to one of three treatment groups. This was communicated to the secretary and hence the physiotherapist and participant.
Six qualified and registered physiotherapists including the primary researcher	To conduct the screening interview on the baseline/ first physiotherapy visit including completion of consent form, health history and demographic questionnaire, painDETECT, lumbar assessment, pain expectation questionnaire, outcome measures, explain pain and activity diaries and pedometer use. Apply treatment throughout the RCT to participants Collect completed pain and activity diaries Help participants with problems encountered in the RCT Ensure completion and collection of outcome measures at six-weeks and 12-weeks follow-up.
Two blinded research assistants	Used to help with capturing data into research database from baseline screening interview, all outcome measures and pain and activity diaries.
RCT statistician	Employed to assist in generating a randomization table, assist in development of data analysis models. Was blinded to the participant allocation of treatment groups.

The main study could commence following booking an appointment to participate in the RCT and arriving at the arranged first physiotherapy visit. All six physiotherapists involved in the RCT including the primary researcher were qualified, physiotherapists, registered with the South African Society of Physiotherapy. The baseline/ first physiotherapy visit of the RCT entailed an order of procedures in Table 30. This first visit included completion of baseline questionnaires, described in Table 23 and 24, undergoing the randomization process and receiving the first treatment. Depending on treatment group allocation, treatments received are briefly described in Table 20. Following the first physiotherapy visit participants were encouraged to book further appointments within the 12-week period. The RCT criteria were designed that there would be a minimum of three physiotherapy visits, and a maximum of nine. The three minimum physiotherapy visits were designed to be arranged at baseline, at six-weeks and at 12-week follow-up in order to capture maximum information from outcome measures. Table 30 outlines which procedures would be conducted in each physiotherapy visit. All data recorded by each physiotherapist, including the outcome from participant examinations and treatment, were stored in a locked cupboard, with access only permitted by the principle researcher and treating physiotherapists. Participants were free to withdraw at any time during the RCT. Participant who requested to withdraw were requested to complete the 12-week follow-up outcome measures.

Table 30: Procedures from baseline to 12-week follow-up.

Physiotherapy visit		Procedures described
Baseline / First physiotherapy visit		Information sheet given, inclusion/ exclusion criteria checked, RCT explained, questions answered Consent form signed Health history and demographic questionnaire completed painDETECT questionnaire completed, scored by physiotherapist and given to practice secretary Participant randomised to one of three treatment groups (through random allocation sequence) Complete form: expectation of pain intensity (PCOQ) Lumbar spine assessment done by physiotherapist Complete forms: outcome measures (NRS, ODI, TSK, PCS) Pedometer explained and provided to participant Pain and activity diary explained and provided to participant Participant receives the first treatment that they have been randomised to Participant books second physiotherapy visit (or later by telephone)
2 3 4 5 6 7 8	Physio visits 2 – 8 (vary depending on discretion of participant and physiotherapist)	Participant received treatment that they have been randomised to Returned any completed pain and activity diary to attending physiotherapist on successive physiotherapy visits (2-9) Participant booked following physiotherapy visit (minimum of three, maximum of nine visits) At the six-week time frame, the participant completed outcome measures (pain intensity, ODI, TSK, PCS) If less than nine visits are received in the 12-week time frame, the participants complete the 12-week/ follow-up outcome measures (pain intensity, ODI, TSK, PCS). If the participant completes the 12-week/ follow-up outcome measures, the participant returns pedometer and any outstanding pain and activity diaries
Physio visit 9 (12-week follow-up)		At the 12-week time frame, the participants completed the 12-week/ follow-up outcome measures (pain intensity, ODI, TSK, PCS). Participant received final treatment that they have been randomised to Participant returned pedometer and any outstanding pain and activity diaries
10		This physiotherapy visit was designed in case participants arrived for the ninth treatment on follow-up and were not able to complete all outcome measures (for collection of fully complete outcome measures)

First physiotherapy visit

The first physiotherapy visit occurred at baseline. Prior to randomization, the physiotherapist assessed each potential participant's eligibility against the inclusion/ exclusion criteria (Table 28), as part of the initial appointment at baseline. This was followed by randomization, baseline data collection as described in tables 23 and 24, and initial treatment. At this appointment, participants were provided with an information sheet (Appendix 15) explaining the nature and purpose of the RCT. The outline of the RCT was then explained, and participants were encouraged to ask further questions, as necessary. After signing the informed consent (Appendix 16), a health history and demographic questionnaire (Appendix 5) would be completed, followed by participants completing a set of baseline outcomes and associated variables (Initial Outcome Measure Questionnaire). This

included the painDETECT questionnaire (Appendix 6). After the pain DETECT questionnaire was completed it was scored by the physiotherapist and given to the practice secretary during the first physiotherapy visit. At this point two processes would occur simultaneously:

1. The practice secretary would call a research assistant off-site with the painDETECT score. This would allow for the simple randomization of the participant by the research assistant. The off-site research assistant used the painDETECT score and which practice the participant was seen at to find the randomization number and treatment type. The off-site research assistant had access to the random allocation sequence list (Appendix 17) and was blinded to the three treatments. Eligible potential participants were randomised using simple randomization from a previously generated computer sequence. Participants were then assigned with equal probability to one of three treatment groups (P, PW, or P). This was communicated back to physiotherapist via the practice secretary.
2. Once treatment group allocation had been determined, an associated baseline variable, measuring expectation of pain by the end of the 12 weeks, was obtained using the Patient Centred Outcomes Questionnaire (Appendix 7). Following this the physiotherapist completed the lumbar assessment (Appendix 8) and proceeded with explaining the nature and purpose of the allocated treatment group. Following this, the participant would then complete the Numerical Rating of Pain Scale (Appendix 9), the Oswestry Disability Index (Appendix 12), the Tampa Scale for Kinesiophobia (Appendix 13), and the Pain Catastrophizing questionnaire (Appendix 14).

Prior to starting the first treatment, participants were provided with instructions for using their pedometer. They were requested to record the outcome from their daily walking, from the pedometer, into their pain and activity diary (Appendix 3 and 4). Participants were encouraged to complete their diary entries daily to maximise accurate monitoring of the steps taken. Diaries were marked with the participants random allocation number. Diaries are described in Table 22.

Depending on group allocation to one of P, PW, W groups, each participant completed the 12-week RCT, described in the intervention planning chapter (Table 20). Due to the subjective nature of pain, not all participants required equal number of treatments. The treatments are briefly compared in the Table 20. Treatments were intended to decrease sensitization and the perception of pain. All the modalities (education on CLBP, massage, manipulation, isometric transversus abdominus stabilization exercise, and pedometer-based walking) were applied by the therapist in order not to sensitize the participant and increase pain intensity. Physical application was based on hand pressures that felt beneficial to the participant and did not illicit pain or increase sensitization. Manipulation was based on Maitland mobilization (Maitland, 2001). It was used according to which type (e.g.:

rotation, postero-antero, unilateral) and grade (I, II, III or IV) of mobilization that relieved the participants pain symptoms. Type and grade of mobilization would be decided by the physiotherapist at each treatment. A systematic review supported sub-maximal isometric transversus abdominus stabilization exercise in CLBP treatment (Gordon and Bloxham, 2016). This evidence-based exercise was used as part of the usual care treatment. The walking intervention too was applied at baseline totalling in weekly minutes < 150 minutes. This minute total ensured the participants started the intervention with a level which was classified as insufficiently active in keeping with modalities justified not to increase sensitization. Furthermore, all participants would receive therapeutic time with a physiotherapist commonly discussing CLBP education and reassuring advice. Additionally, every treatment group had an exercise component. The PW treatment group had both exercises in one treatment group (isometric lumbar stabilization and a pedometer-based walking intervention). All questionnaires and outcome measures completed at baseline are listed in Table 23 and 24.

Physiotherapy visits two-nine

Following the first physiotherapy visit, visits for treatment would be booked prospectively after attending each treatment. This would be done by telephone, with the practice secretary to ensure consistency with the same treating physiotherapist, for the duration of the RCT. This treatment booking approach was consistent with that used in private physiotherapy practices in South Africa. Physiotherapy visit number two-nine would aim to replicate the initial treatment. Participants self-selected further physiotherapy visits. Consequently, the number of physiotherapy visits per participant varied, with not all participants accessing the nine available physiotherapy appointments. These treatments were spread over the 12-week intervention period.

Pain and activity diaries completed during the weeks prior to the appointment, were collected by the physiotherapist. These were and then stored in a locked cupboard, with only the principal researcher having access to the cupboard.

Repeated outcome measures

Follow-up visits to include repeated outcome measures were scheduled by the treating physiotherapist' at six-week (Appendix 10, 12, 13, 14) and 12-week (Appendix 11, 12, 13and 14) follow-up visits. The repeated outcome measures, NRS, ODI, TSK and PCS were conducted as per baseline procedures. The treating physiotherapist would provide the outcome measure forms and assist the participant in their completion. If the participants were not able to complete the outcomes or forgot their diaries at the ninth treatment, a tenth physiotherapy visit would be arranged for maximum data collection. Nine and ten physiotherapy visits were categorized as the same category in number of physiotherapy visits.

Methods to control bias

The RCT was conducted in accordance with the CONSORT checklist (Moher et al., 2010). The RCT design includes the methodological feature of true randomisation which is recognised as minimising bias in clinical trials. Participants would be randomised to three treatment groups by a predetermined allocation schedule and not based on the researcher's judgement. Specification of eligibility criteria and intention to treat are met through similar CLBP walking intervention studies to obtain maximum information for participants with CLBP. Physiotherapists involved in this RCT worked equally at all three sites and received standardized training on assessment and treatment protocols, reducing bias and ensuring consistency of treatment and assessment. Assessment and treatment notes were reviewed regularly by the principal investigator to address any deviation from the protocol. Participants and physiotherapists were instructed not to talk to other patients/ participants who may be potentially included in the RCT to maintain blinding. Participants were asked not to receive any other treatment or start any new form of exercise other than the one they had been randomised to during the RCT period. Participants were reminded of their physiotherapy appointments and their six and 12-week-follow-up outcome measure evaluations after each successive treatment, or by telephone if a participant missed a treatment. Participants were furthermore reminded not to reveal their treatment to people wishing to participate in the RCT.

3.10 Incentive

All participants were offered a pharmacy gift voucher upon trial participation.

3.11 Data management and analysis

A statistician was employed to generate a random allocation sequence and assist with data analysis. The statistician was not involved in collection of data or delivery of treatment. Two research assistants remained blinded to the RCT group allocation.

Sample size

G*Power was used to calculate sample size (Faul et al., 2007). Sample size estimation was based on a between-group repeated measures analysis of (a) weekly average pain measurements on a 100-mm VAS scale and (b) baseline, 6-week, and 12-week measurements of the ODI on a scale of 0-100%. The estimations are based on a 5% significance level, 80% power and a 0.5 correlation among the repeated measures.

Sample size was calculated based on a minimum clinically relevant between-group difference in pain intensity measurement of 10mm/100mm on a VAS scale, using a standard deviation of 25mm

(Torstensen et al., 1998; Mirovsky et al., 2006). Estimated Sample size calculated was 147 participants. At the time of sample size calculation only one systematic review had been done on using walking as a treatment for CLBP (Hendrick et al., 2010).

The minimum clinically relevant between-group difference used in ODI is 10% (Hägg and Fritzell, 2003; Bombardier, Hayden and Beaton, 2001; Ostelo and de Wet., 2005). The within-group standard deviation was 15 (Torstensen et al., 1998; Mirovsky et al., 2006). The estimated sample size for using ODI as an outcome measure was 72.

To include both NRS for pain and ODI, the larger sample size of 147 was required for this RCT (50 for each treatment arm was used for convenience). This RCT was powered to detect between-group repeated measures of pain intensity and ODI but not differences between groups in nociceptive and neuropathic pain phenotypes.

Randomisation

The RCT statistician used a randomization table (random allocation sequence) which was generated using the RCT statistician's random computer software. Simple randomization was used to maintain complete randomness of the assignment of participants to one of three treatment groups. The random allocation sequence was kept in a locked cupboard offsite.

Data analysis

The RCT was analysed on an intention-to-treat basis. All data were tabulated by treatment group (P, W and PW). For those participants whose treatment included a daily walking intervention (W and PW), daily number of steps, distance walked (km), and duration (minutes) was averaged for each week of the 12-weeks of the RCT. All data were coded and entered as continuous or categorical data. A data analysis plan was prepared and given to the independent RCT statistician.

The coding of baseline assessments was done on Microsoft Excel by two independent research assistants. After the 12-weeks of treatment, information from the pain and activity diaries was coded on Microsoft Excel together with the remaining outcome measures (6-week and 12-week follow-up) by one research assistant and the principal researcher who remained blinded, having access only to the anonymous details of the participants. Data analysis was conducted using SAS version 9.4. The 5% significance level was used throughout.

Parametric and non-parametric statistical tests were selected after examination of the residual distributions. If the assumptions of normality were not met, non-parametric tests were used.

Descriptive analysis of the data was conducted as follows:

- Categorical variables were summarised by frequency and percentage and illustrated by means using a bar chart.
- Continuous parametric variables were summarised by mean and standard deviation.
- Continuous non-parametric data as median and interquartile range.
- Continuous variables were illustrated by means of box-and-whisker plots.

Outcome data were presented in histograms and examined for distribution and outliers.

The number of weeks each participant spent in the RCT was determined from the last week in which a total step count was recorded in the pain and activity diary. Missing outcome data were replaced by the corresponding baseline observation (week 1 observations in the case of walking measurements).

Participant dropout was analysed. Association between number of weeks in the RCT and treatment group was analysed using a Chi-Squared test. The effect of pain phenotype on drop out was analysed using a Fishers exact test. The effect of treatment, pain phenotype, baseline outcome on dropout (categories included: less than 12-weeks in RCT vs. 12-weeks in RCT) for each of the Pain Intensity (NRS), ODI, TSK and PCS outcomes was determined using logistic regression with dropout as the dependent variable, and outcome at baseline, treatment group, and pain phenotype as independent variables. The effect of last measured outcome on dropout, treatment group, and pain phenotype was determined similarly. In the dropout analysis nociceptive pain phenotype and PW treatment group were chosen as the reference categories since these had the lowest dropout. Only main effects were assessed because sample size considerations for logistic regression estimated that at most b = 4 parameters could be included in the model based on the smallest outcome class having 10*b cases (dropouts = 40, and hence b = 4 parameters (Peduzzi et al 1996).

Categorical variables were compared using the X^2 test. Fisher's exact test was used for 2 x 2 tables or where the requirements for the X^2 test could not be met. Continuous variables were compared using the independent samples t-test (or the Wilcoxon rank sum test where the assumptions of the t-test were not met). The strength of the associations was measured by the Cohen's d for parametric tests and the r-value for the non-parametric tests. Hypothesis tests were used. The strength of the associations was measured by Cramer's V and the phi coefficient, respectively. The scale of interpretation for these effect sizes is shown in Table 31.

Table 31: Interpretation of effect size measures

X^2 test/ Fisher's exact test		Independent samples t-test / Wilcoxon rank sum test	
Cramer's V / phi coefficient	**Interpretation**	**Cohen's d / r-value**	**Interpretation**
0.50 and above	High/strong association	0.80 and above	Large effect
0.30 to 0.49	Moderate association	0.50 to 0.79	Moderate effect
0.10 to 0.29	Weak association	0.20 to 0.49	Small effect
Below 0.10	Little if any association	Below 0.20	Near zero effect

Baseline variables were compared between treatment groups using standardised mean effect size (SMES) (Woodward 2013). Where some studies demonstrate the statistical significance, which is the probability that the observed difference between two groups is due to chance, the effect size is independent of sample size. Typically, participants in the groups in an RCT are 'the same' in the population at large, and hypothesis testing will only be as effective as the randomisation process used; the larger the sample size, and the more comparisons made, the more likely we were to obtain at least one significant result. In this RCT, differences in the cohort were examined using SMES.

Normally a threshold of 10% for important imbalance between groups is used but given the small sample size (which increases the risk of imbalance), focus was on the differences > 30%. In summary tables, the magnitude of the SMES was categorized: 10-19%; 20-29%; and 30% or more.

The number of physiotherapy visits was compared by treatment group using a Chi-Squared test.

The outcome measure analyses used in this RCT are demonstrated in Table 32. Analysis was performed in an order of sequence. If an association with predictive variables occurred during modelling, more than one model would be generated. Pain phenotype was used in the modelling process.

Table 32: Sequence of outcome measure analysis presentation

Outcome measure	Analyses	Models generated
Pain Intensity (NRS), ODI, TSK, PCS	Preliminary data analysis. Summary measures for mean NRS, ODI, TSK, and PCS. Mean change in score from week 0 calculated for week 6 and 12	None
Pain Intensity (NRS)	ANOVA	
	Primary outcome measure analysis: Primary analysis using LMM	1 and 2
	Primary outcome measure analysis: Investigative analysis using LMM	3 and 4
ODI	ANOVA	
	Secondary outcome measure analysis using LMM	5 and 6
TSK	ANOVA	
	Secondary outcome measure analysis using LMM	7
PCS	ANOVA	
	Secondary outcome measure analysis using LMM	8

ANOVA: Analysis of variance (baseline-week-12 follow-up: did not control for any independent variables)

LMM: Linear mixed model (baseline, six and 12-week follow-up)

As a preliminary analysis of the data, two summary measures were calculated. Firstly, the mean (95% confidence interval) for the NRS, PCS, TSK, and ODI for each treatment group (P, PW, W) at each time-period (weeks 0, 6 and 12) was calculated. Then, for each intervention, the mean (95% confidence interval) change in score from week 0 was calculated for week 6 and 12 for each outcome measure. Finally, to assess the effect size between the mean change score at each time interval, Cohen's *d* values were calculated for P and PW versus W for each outcome measure. Cohen suggested that $d = 0.2$ be considered a small effect size, $d = 0.5$ be considered a 'medium' effect size, and $d = 0.8$ be considered a large effect size (Cohen, 1988).

The primary analysis was conducted on the primary outcome measure which was pain intensity using an analysis of variance (ANOVA) and an analysis using Linear mixed models (LMM) (Table 32). Pain intensity was used as the dependant variable. In the primary analysis, potential predictors of pain intensity at 12-week follow-up included the independent variables: pain intensity at baseline, treatment group allocation (P, W, or PW), number of physiotherapy visits and pain phenotype. These independent variables were selected a priori, based on the literature. The ANOVA was used to determine a statistically significant difference in pain intensity score from baseline to 12-week follow-up between groups. The independent variables' effect on pain intensity (NRS) at 12-week follow-up was determined using a LMM. Prior to the analyses, the association between each pair of independent variables was assessed to identify any strong associations which could introduce confounding effects into the model.

Following the primary analysis, an investigative analysis of pain intensity was conducted using linear mixed models (Table 32). Standard modelling was used. Factors commonly associated with pain intensity were included in the investigative modelling (Meucci et al 2015, Dionne et al 2001, Watson et al 2004, Dionne et al 2006, Shiri et al 2010). Factors comprised of:

- age
- gender
- employment
- education
- smoking status
- BMI

In the analysis, where time is used as an independent variable in the analysis, two time points were used: six-week follow-up and 12-week follow-up. The effect of treatment, time in intervention, pain phenotype, baseline NRS and selected covariates on the pain intensity outcome measure (NRS) was determined using a LMM, with the NRS as the dependent variable, and NRS at baseline, treatment group allocation, time (week in the RCT), treatment group allocation x time interaction, pain phenotype, pain phenotype x treatment group allocation interaction, pain phenotype x time interaction, and the other covariates as independent variables. The participant was the random effect in the linear mixed model.

For all secondary outcome measures of disability, kinesiophobia and pain catastrophizing, a secondary outcome measure analysis was conducted using an ANOVA and linear mixed models (Table 32). The analyses were conducted on each outcome measure (ODI, TSK, and PCS) separately. In each secondary outcome measure analysis, the outcome measured, either ODI, TSK or PCS, was classified as the dependant variable. For each outcome measure, an ANOVA was used to determine a statistically significant difference in secondary outcome measure score from baseline to 12-week follow-up between groups at 12-week follow-up. In the LMM for each secondary outcome measure, as with the investigative analysis of pain intensity, standard modelling was used. The same factors commonly associated with pain intensity were included in the modelling and comprised: age, gender, employment, education, smoking status, and BMI (Meucci et al 2015, Dionne et al 2001, Watson et al 2004, Shiri et al 2010).

The effect of treatment group allocation, time, pain phenotype, baseline outcome and selected covariates on ODI, TSK, and PCS outcome measures was determined using a LMM (Table 32) with the outcome as the dependent variable, and outcome measure at baseline, treatment group allocation, time (week in the RCT), treatment group allocation x time interaction, pain phenotype, pain phenotype x treatment group allocation interaction, pain phenotype x time interaction, and the other

covariates as independent variables. In the analysis, where time is used as an independent variable in the analysis, two time points were used: six-week follow-up and 12-week follow-up. The participant was the repeated measure.

Prior to these analyses, the association between each pair of independent variables was assessed to identify any strong associations which could introduce confounding effects into the modelling. The association between a continuous and a categorical variable was assessed by the independent samples t-test (or one-way ANOVA in the case of more than two categories). Non-parametric equivalents (the Wilcoxon rank sum and the Kruskal-Wallis tests, respectively) were used where necessary. The association between two categorical variables was assessed by the X^2 test, or Fisher's exact test was used for 2 x 2 tables or where the requirements for the X^2 test could not be met. Finally, the association between two continuous variables was assessed by Pearson's correlation coefficient (or Spearman's correlation coefficient where the assumptions of the former test could not be met).

The strength of the associations was measured by the Cohen's d for parametric tests and the r-value for the non-parametric tests.

In additional analysis, the relationship between expectation of pain intensity (following the 12-week RCT) and the actual change in pain intensity was investigated. The correlation between expectation of pain intensity at baseline and the change in pain between baseline and 12-weeks was assessed using Spearman's rank correlation coefficient (since some of the data were not normally distributed).

An independent research assistant was instructed to process the walking data from the pain and activity diaries. To account for missing PA data, steps were averaged for each week. Weekly step count was averaged using total number of steps taken over a week, divided by the number of days that steps were recorded. The variation in the number of days recorded per week, were not considered. This method was used for walking done as PA of total daily steps and for the advised walking that was performed in the walking intervention. Diaries that had a minimum of one day per week through to seven days per week of steps recorded were included. The number of weekly steps taken, and weekly minutes spent performing the walking intervention were presented as means. The ideal walking program in weekly minutes was plotted against the two walking interventions. The descriptive statistics for all the walking variables are presented by treatment group as mean, standard deviation, and weekly mean steps divided by seven for interpretation as average steps per day. The effect of treatment group allocation, time (week in the RCT) and pain phenotype on total weekly steps was determined using a LMM with total weekly steps as the dependent variable, and treatment group allocation, time, and pain phenotype as independent variables.

Chapter 4: Results

4.1 Study recruitment and randomisation

Recruitment commenced in August 2015 and continued until the achievement of recruitment targets, in March 2017. Random assignment ensured no bias toward group allocation.

Five hundred and forty patients were assessed for eligibility, from which 153 were randomised to intervention or control conditions. Participants were randomised to one of three treatment groups:

- Usual Care/ Physiotherapy (P. n = 48)
- Pedometer-based walking intervention (W. n=54)
- Usual care/ Physiotherapy combined with a Pedometer-based walking intervention (PW. n=51).

This was a 12-week intervention, with repeated measures at 6-weeks and a final follow-up at 12-weeks. The CONSORT flow chart describing participant flow is shown in figure 9.

Figure 9: The flow of participants through the RCT, in-line with the CONSORT statement (Schulz et al 2010).

4.2 Participant dropout

This section will present results relating to attrition during the RCT. Dropout associated with either treatment group or pain phenotype was assessed. Pain intensity associated with dropout was assessed as this was the primary outcome measure.

Participant dropout characteristics

Table 33 describes dropout by week of intervention. In total, 27% (n=40) of participants had dropped out by 12-week follow-up.

Table 33: Drop out, by week of intervention

RCT week	Drop out	
	N	%
	147	100
1	17	11.6
2	6	4.1
3	5	3.4
4	3	2.0
5	1	0.7
6	4	2.7
7	3	2.0
11	1	0.7
12	0	0

To demonstrate the number of participants who completed the RCT, participants were categorized into two groups, not completing the 12-week intervention, and having completed the 12-week intervention. Dropout was compared by treatment group (Table 34) and by pain phenotype (Table 35 and 36).

A chi-squared test demonstrated a statistically significant association between treatment group and drop out. The PW treatment group were more likely to stay in the RCT compared to the other two treatment groups (p=0.02); however, the effect size of this was weak (Cramer's V=0.23) (Table 34).

Table 34: Participants completing the 12-week RCT or dropping out before by treatment group.

		Entire cohort n (%)	P n (%)	W n (%)	PW n (%)	p value
Number of weeks in the study	<12	40(27.2)	13(28.3)	20(38.5)	7(14.3)	0.02*
	12	107(72.8)	33(71.7)	32(61.5)	42(85.7)	

Groups compared with Chi² n (%)

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

A fisher's exact test demonstrated no association between pain phenotype and drop out (p=0.09).

Dropout and the effect of pain intensity

Binary logistic regression was used explore whether baseline pain intensity scores were a potential predicator of participant drop out. The dependant variable (drop out) was categorical with one of two responses predicting drop out; pain intensity at baseline in participants that did complete the 12-week RCT, and pain intensity at baseline in those who did not complete the 12-week RCT. Independent variables entered into the equation were baseline pain intensity, allocation to treatment group and pain phenotype (Table 35). After controlling for all other variables in the model, there was a significant effect for pain intensity score at baseline, on drop out, in the W treatment group compared to the PW treatment group (p=0.01, OR 4.18, 95% confidence intervals (CI) 1.54-11.37). Moreover, after controlling for the other variables in the model (pain intensity at baseline, and allocation to treatment groups), there was an effect of pain phenotype on drop out. The odds of dropout were higher for the neuropathic pain phenotype compared to the nociceptive pain phenotype (p=0.04, OR 2.55 (95% CI 1.04 to 6.22)).

Table 35: The association between participant dropout and pain intensity at baseline, treatment group allocation, and pain phenotype.

Effect	p value	Odds ratio	95% Confidence intervals for odds ratio	
			Lower	Upper
Pain intensity at baseline:	0.59	0.99	0.97	1.02
Treatment: P vs. PW	0.10	2.40	0.85	6.79
Treatment: W vs. PW	0.01**	4.18	1.54	11.37
Pain phenotype: Neuropathic vs. Nociceptive	0.04*	2.55	1.04	6.22

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Binary logistic regression was used explore whether last observed pain intensity scores were a potential predicator of participant drop out. Independent variables entered into the equation were last

observed pain intensity, allocation to treatment group and pain phenotype (Table 36). After controlling for all other variables in the model (allocation to treatment group and pain phenotype), there was a significant effect of last observed pain intensity score on drop out (p=0.01, OR 1.03 (95% CI 1.01to1.04)).

Moreover, after controlling for the other variables in the model (last observed pain intensity, and allocation to treatment group (P vs. PW), pain phenotype), there was a significant effect of last observed pain intensity score on drop out, in the W treatment group compared to the PW treatment group (p=0.01, OR 3.81 (95% CI 1.33 to 10.91)).

Table 36: The association between participant dropout and last observed pain intensity, treatment group allocation, and pain phenotype

Effect	p value	Odds ratio	95% Confidence Limits for odds ratio	
			Lower	Upper
Pain intensity last observed:	0.01***	1.03	1.01	1.04
Treatment: P vs. PW	0.08	2.67	0.89	8.01
Treatment: W vs. PW	0.01*	3.81	1.33	10.91
Pain phenotype: Neuropathic vs. Nociceptive	0.33	1.51	0.66	3.48

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

4.3 Baseline characteristics

Baseline characteristics by treatment group are described below. There were no missing data in this section.

Baseline characteristics of treatment groups

The RCT's primary outcome measure was pain intensity. In order to detect a 10-point difference on a 100-point pain scale, assuming a 25-point standard deviation, at 80% power required a sample size of 147 participants. A total of 153 participants consented to the RCT however six participants were withdrawn prior to completing the baseline interview due to exclusion criteria, see figure 9. Baseline characteristics are described in Table 37.

Table 37: Baseline characteristics by treatment group

		Entire cohort	P	W	PW	Standardized mean effect sizes between treatment groups in the effect of the intervention versus usual care.	
						P/PW	P/W
Age (years)*		46.2 (10.9)	45.4 (9.6)	47.7 (11.3)	45.5 (11.6)	0.3%	12.1%
Gender Female. n (%)		92 (62.6%)	27 (58.7%)	35 (67.3%)	30 (61.2%)	5.2%	17.9%
Gender Male. n (%)		55 (37.4%)	19 (41.3%)	17 (32.7%)	19 (38.8%)	5.2%	17.9%
BMI (kg/m^2) **		27.3 (24.7-30.1)	26.7 (25.1-28.6)	27.5 (24.4-32.2)	27.4 (24.2-30.3)	3.2%	6.3%
Highest education achieved	Completed high school. n (%)	60 (40.8%)	16 (10.9%)	18 (12.2%)	26 (17.7%)	4.1%	31.2%
	Diploma. n (%)	45 (30.6%)	16 (10.9%)	12 (8.1%)	17 (11.6%)	0.2%	26%
	Degree. n (%)	42 (28.6%)	14 (9.5%)	14 (9.5%)	14 (9.5%)	4.1%	7.8%
Smoker. n (%)		58 (39.5%)	19 (41.3%)	18 (34.6%)	21 (42.9%)	3.1%	13.8%
Employed. n (%)		135 (91.8%)	45 (97.8%)	47 (90.4%)	43 (87.8%)	39.7%	32%
Pain duration (years)**		8 (4-13)	8 (5-10)	10 (5-14)	7 (3-14)	6.9%	6%
Neuropathic. n (%)		70 (47.6%)	23 (32.9%)	22 (31.4%)	25 (35.7%)	2.4%	15.5%
Nociceptive. n (%)		77 (52.4%)	23 (29.9%)	30 (39.0%)	24 (31.2%)	2.4%	15.5%
Expectation of Pain Intensity after RCT at 12-weeks *		16.9(18.4)	15.8(14.9)	19(19.9)	15.6(19.8)	9.8%	4.2%
Pain Intensity *		53.9(21.8)	52.3(21.7)	53.0(20.7)	56.5(23.3)	18.9%	3.2%
Oswestry Disability Index *		24.9(11.2)	26.7(12.2)	25.0(11.0)	23.3(10.4)	12.4%	5.2%
Tampa Scale for Kinesiophobia *		38.4 (7.1)	39.7(6.1)	38.1(8.2)	37.6(6.6)	33.8%	21.9%
Pain Catastrophizing Scale *		17.9(12.2)	16.6(11.9)	19.8(12.3)	17.0(12.2)	0.6%	12.7%

P, usual care treatment; **W,** pedometer-based walking intervention;

PW, usual care treatment and pedometer-based walking intervention

*mean (SD) **median (IQR)

Numerical Rating Scale (0-100): higher score indicates more pain intensity

Oswestry Disability Index (0-100): higher score indicates more LBP-related functional disability

Tampa Scale of Kinesiophobia (17-68): Scores ≥37 indicates more fear avoidance beliefs

Pain Catastrophizing Scale (0-52): Scores ≥30 indicates more pain catastrophizing behaviour

SD: Standard deviation

IQR Interquartile range

4.4 Physiotherapy visits

The numbers of physiotherapy visits made by participants in each treatment group, over the 12-week intervention, are described in Table 38. There were no missing data in this section.

Table 38: Number of physiotherapy visits in the study

Total number of physiotherapy visits	**Entire cohort n (%)**	**P n (%)**	**W n (%)**	**PW n (%)**	**p value ***
1-2	28(19.1)	6(13.0)	19(36.5)	3(6.1)	
3-4	34(23.1)	7(15.2)	19(36.5)	8(16.3)	
5-6	31(21.1)	10(21.7)	8(15.4)	13(26.5)	**<0.01**
7-8	29(19.7)	13(28.3)	3(5.8)	13(26.5)	
9-10	25(17.0)	10(21.7)	3(5.8)	12(24.5)	

*Groups compared with Chi² n (%)

To capture the follow-up information, all participants were asked to book a minimum of three physiotherapy appointments. These were included at:

- Baseline
- Six-week follow-up
- 12-week follow-up

Table 38 demonstrates the number of physiotherapy visits varied between participants with 28 participants attending less than three physiotherapy visits.

4.5 Outcome measures

Outcome measures of Pain Intensity, Disability, Kinesiophobia and Catastrophizing were measured in all participants (n-147), at:

- Baseline (0 weeks)
- Six-week follow-up
- 12-week follow-up

Pain intensity (NRS), disability (ODI), kinesiophobia (TSK), and pain catastrophizing (PCS) data were described with mean and standard deviation. Outcome measure data observed at baseline was normally distributed. There were no missing data in this section.

To track the four outcome measures from baseline to follow-up at 12 weeks, summary statistics were given in table 39 and table 40.

Table 39. Summary statistics for the four outcome measures at each time point for the three treatment groups

Treatment group Mean change (95% confidence interval)			
	P	**PW**	**W**
Week 0	52.3 (45.9 to 58.7)	56.5 (49.8 to 63.2)	53 (47.2 to 58.7)
Week 6	37.1 (29.9 to 44.3)	36.6 (29.6 to 43.6)	42.1 (34.8 to 49.4)
Week 12	33.5 (26 to 41)	29.6 (21 to 38.2)	40.2 (32.7 to 47.7)
ODI (% scale, where 0% indicates no disability)			
Week 0	26.7 (23.1 to 30.3)	23.3 (20.3 to 26.3)	25 (21.9 to 28)
Week 6	20.7 (17.1 to 24.4)	18 (14.7 to 21.2)	22.7 (19.2 to 26.1)
Week 12	19 (15.1 to 23)	14.6 (11.2 to 18)	19.8 (16.6 to 23)
TSK (17 to 69 scale, where 17 indicates no kinesiophobia)			
Week 0	39.7 (37.9 to 41.5)	37.6 (35.7 to 39.5)	38.1 (35.8 to 40.4)
Week 6	36.2 (33.3 to 39.1)	33 (31 to 34.9)	35.6 (33.6 to 37.6)
Week 12	36.7 (34.4 to 39)	31.5 (29 to 34)	34.8 (32.6 to 37)
PCS (0 to 52 scale, where 0 indicates no catastrophizing)			
Week 0	16.6 (13.1 to 20.2)	17 (13.5 to 20.5)	19.8 (16.3 to 23.2)
Week 6	12.1 (8.58 to 15.7)	9.65 (6.83 to 12.5)	15.2 (11.9 to 18.5)
Week 12	11.3 (7.94 to 14.8)	9.04 (5.82 to 12.3)	14.7 (11.2 to 18.1)

Mean changes from baseline at both follow-up points showing reductions in scores in all three interventions and improvements in all three interventions. With a minimally clinically important difference in pain intensity of >2/10, the PW intervention showed a MCID at 12-week follow-up. All three intervention groups showed high degrees of kinesiophobia and all three intervention groups showed levels of kinesiophobia lower than the cut-off score of 37 at both time points. The largest changes in mean TSK score were seen in the PW treatment group.

Table 40. Mean change from baseline scores for all outcome measures and between-group effect sizes*

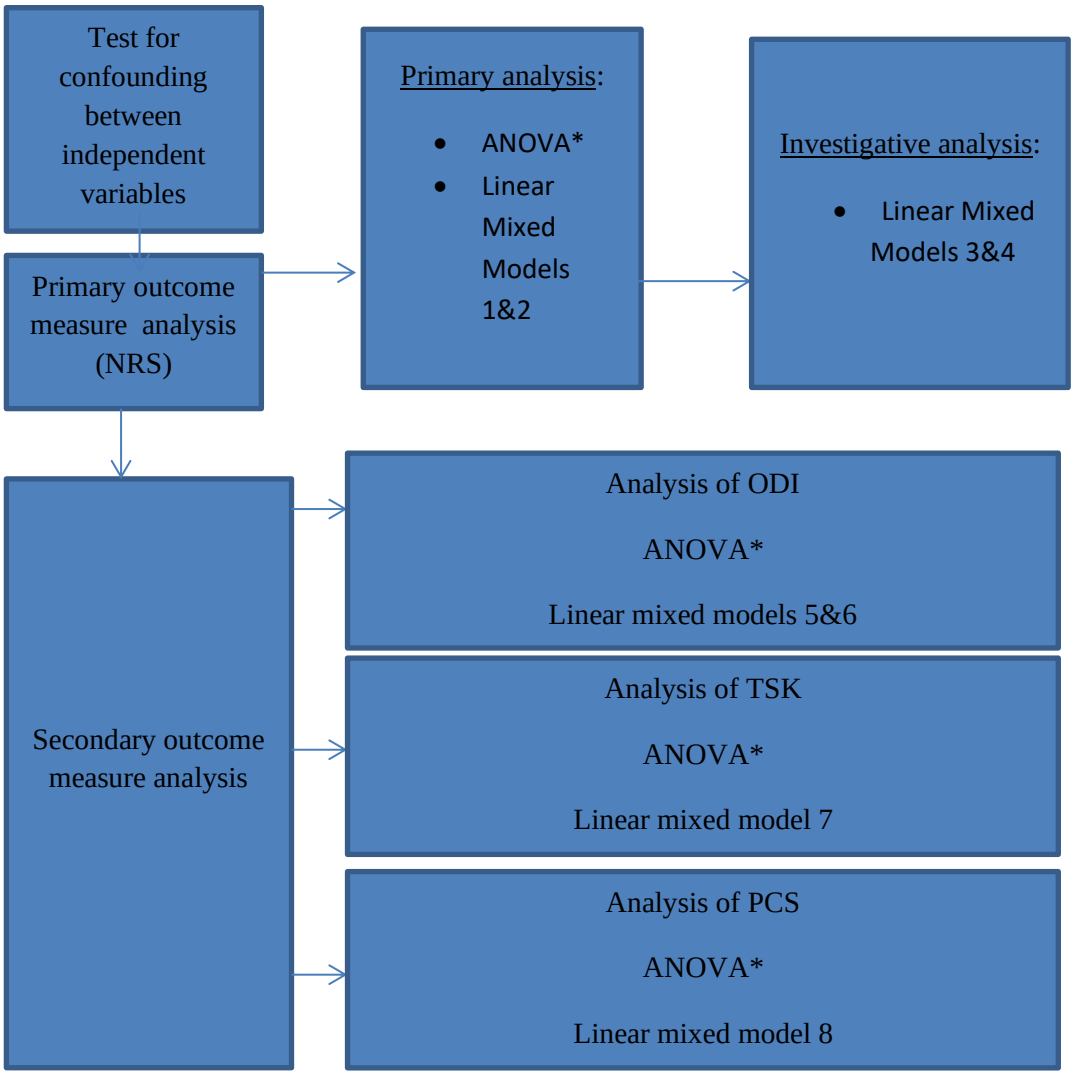
	Treatment group Mean change (95% confidence interval)		
	P	**PW**	**W**
NRS (0 to 100 scale, where 0 indicates no pain)			
Week 6	-15.2 (-22.5 to -7.87)	-19.9 (-27.9 to -11.9)	-10.9 (-16.3 to -5.47)
Cohen's *d*	-0.2	-0.38	REF
Week 12	-18.8 (-26.3 to -11.2)	-26.9 (-35.9 to -17.9)	-12.8 (-19.8 to -5.74)
Cohen's *d*	-0.24	-0.5	REF
ODI (% scale, where 0% indicates no disability)			
Week 6	-5.93 (-8.3 to -3.57)	-5.31 (-8.07 to -2.54)	-2.31 (-4.65 to 0.03)
Cohen's *d*	-0.44	-0.33	REF
Week 12	-7.63 (-10.4 to -4.85)	-8.67 (-11.8 to -5.51)	-5.15 (-7.72 to -2.59)
Cohen's *d*	-0.27	-0.35	REF
TSK (17 to 69 scale, where 17 indicates no kinesiophobia)			
Week 6	-3.5 (-5.31 to -1.69)	-4.61 (-6.51 to -2.71)	-2.5 (-4.38 to -0.62)
Cohen's *d*	-0.16	-0.32	REF
Week 12	-2.98 (-4.44 to -1.51)	-6.06 (-8.14 to -3.99)	-3.27 (-5.37 to -1.17)
Cohen's *d*	0.05	-0.38	REF
PCS (0 to 52 scale, where 0 indicates no catastrophizing)			
Week 6	-4.48 (-6.87 to -2.09)	-7.37 (-10.1 to -4.61)	-4.56 (-7.16 to -1.96)
Cohen's *d*	0.01	-0.3	REF
Week 12	-5.26 (-7.71 to -2.81)	-7.98 (-10.9 to -5.07)	-5.1 (-8.08 to -2.12)
Cohen's *d*	-0.02	-0.28	REF

* Between-group effect sizes: P vs W (reference group) and PW vs W (reference group)

Outcome measures are presented as independent sections and will be reported in the following sequence (figure 10):

- Primary outcome measure analysis (ANOVA and linear mixed model regressions analyses): NRS
- Secondary outcome measure analysis (ANOVA and linear mixed model regressions analyses): ODI, TSK and PCS

Figure 10: Sequence of outcome measure analyses:



*ANOVA (baseline - 12-week follow-up: did not control for any independent variables)
Linear mixed model (baseline, 6-week follow-up, 12-week follow-up)

Association between independent variables

Association was examined between predictive variables as described in the data analysis section of the methods. Where there was statistically significant association between predictive variables, only one of these variables was used in modelling associations with the primary outcome measure and secondary outcome measures.

Exploration for the confounding variables for outcome measures at 12-week follow-up in the models used, included baseline data (Table 41). The association between each pair of covariates was assessed to identify any strong associations which could introduce a confounder into the modelling.

Table 41: A summary of the independent variables included in the models for each outcome measure (NRS, ODI, TSK, and PCS)

Covariate	NRS	ODI	PCS	TSK
Age	√	√	√	√
Gender	√	√	√	√
Employment level	√	√	√	√
Education level	√	√	√	√
Smoking	√	√	√	√
BMI	√	√	√	√
# of Physiotherapy visits	√		√	√
Pain intensity at 12-week follow-up		√		
Pain duration (years)	√	√	√	√
Pain intensity at baseline	√			
ODI score at baseline		√		
PCS at baseline			√	
TKS at baseline				√
Pain phenotype	√	√	√	√

NRS: Numerical Rating Scale for Pain Intensity

ODI: Oswestry Disability Index

TSK: Tampa Scale of Kinesiophobia

PCS: Pain Catastrophizing Scale

√: indicates an assumption that the predictors are independent of each other. If associations were found between above predictors, in which case the predictor was excluded from the model.

The following confounded pairs of variables were identified (Table 41); the strength of association was measured using Cohen's d, where values greater than 0.8 are considered as large:

- Pain phenotype and pain intensity score at baseline (Cohen's d=1.1)
- Pain phenotype and ODI score at baseline (Cohen's d=1.1)
- Pain intensity at 12-weeks and number of physiotherapy visits (Cohen's d=0.9)

Therefore, the following independent variables (Table 41) could not be used together in the linear mixed model:

- For primary outcome measure analysis (NRS): Pain intensity at baseline and pain phenotype.
- For the secondary outcome measure analysis for ODI: ODI at baseline and pain phenotype; number of physiotherapy visits and pain intensity at 12-week follow-up.
- For the secondary outcome measure analysis for TSK: The number of physiotherapy visits and pain intensity at 12-week follow-up.
- For the secondary outcome measure analysis for PCS: The number of physiotherapy visits and pain intensity at 12-week follow-up.

Due to pain phenotype having a strong association with both NRS at baseline and ODI at baseline, this led to the development of two models for NRS and again for ODI outcomes at 12-week follow-up. Therefore, in the primary analyses and investigative analysis of the primary outcome measure analysis where the dependant variable was the NRS (Figure 10), two models were constructed. Models 1 and 2 for the primary analysis, and models 3 and 4 for the investigative analysis:
Model 1: Over and above other predictive variables the model controlled for baseline pain intensity score but excluded pain phenotype, and
Model 2: Included pain phenotype but excluded baseline pain intensity score.
Model 3: Included all the predictive variables in Table 41. Over and above other predictive variables the model controlled for baseline pain intensity score but excluded pain phenotype, and
Model 4: Included all the predictive variables in Table 41. Included pain phenotype and excluded baseline pain intensity score.

In the secondary outcomes analyses where the dependant variable was the ODI, two models were constructed (Models 5&6):
Model 5: Over and above other predictive variables the model controlled for baseline ODI score and excluded pain phenotype, and
Model 6: Over and above other predictive variables the model controlled for pain phenotype and excluded baseline ODI score.
For these models that used ODI as a dependant variable, number of physiotherapy visits was a confounding variable and was excluded from the model.

In the two secondary outcome measures analyses where the dependant variables were the TSK and PCS respectively, only one model was required for each of the TSK (Model 7) and PCS (Model 8) outcomes. Pain phenotype did not act as a confounding variable with either of these secondary outcome measures. Independent variables which were confounding variables with the TSK and PCS had to be excluded from the models. For the models where TSK and PCS scores at 12-week follow-up were the dependant variables, the independent variable of pain intensity at 12-week follow-up was excluded.

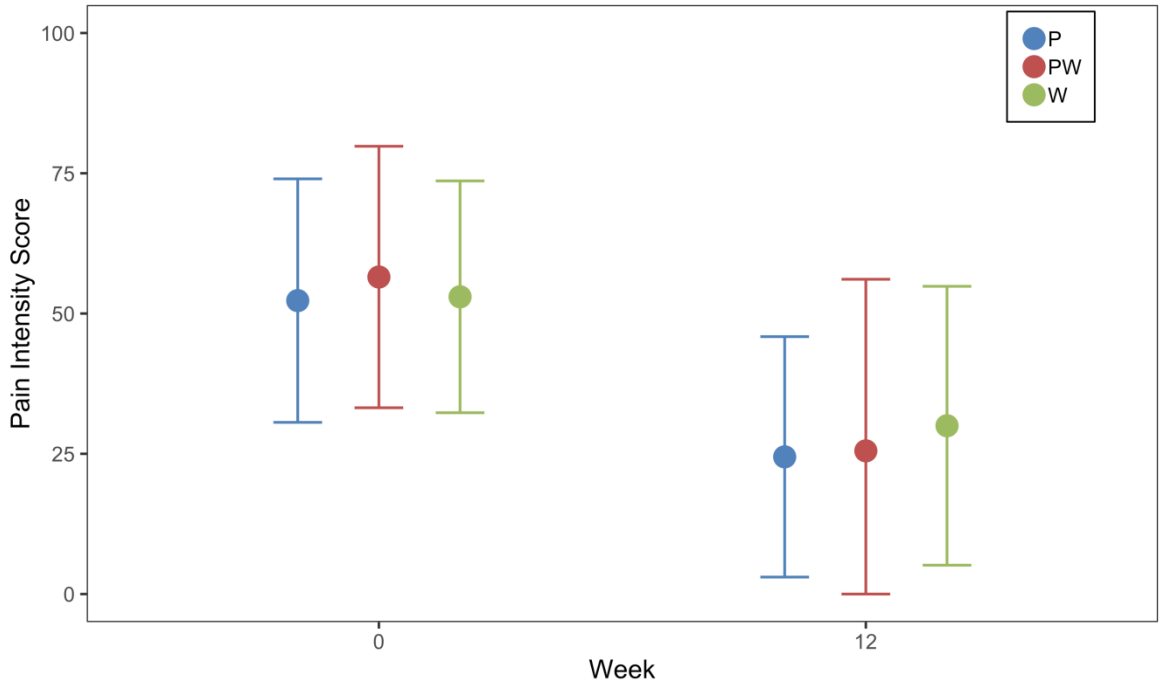
Primary outcome measure analyses

The primary analyses investigated whether there was a significant difference in pain intensity between treatment groups at 12-week follow-up using analysis of variance (ANOVA) and two linear mixed models. An investigative analysis of the primary outcome was also conducted to determine if any of the independent variables referred to in Table 41 included in the models predicted pain intensity at 12-week follow-up using two linear mixed models.

Primary analysis of pain intensity

An ANOVA was used to determine any differences in pain intensity between baseline and 12-week follow-up between treatment groups. There was no statistically significant difference in the change in pain intensity scores between baseline and 12-week follow-up between treatment groups ($F(2,103) = 1.33$, $p = 0.27$; Figure 11)

Figure 11: Change in pain intensity between treatment groups from baseline to 12-week follow-up.



P, usual care treatment; **W,** pedometer-based walking intervention;
PW, usual care treatment and pedometer-based walking intervention;
Week 0, baseline; **Week 12**, 12-week follow-up

Due to baseline pain intensity and pain phenotype being confounding variables, results were presented in Models 1 and 2. A linear mixed model regression analysis compared pain intensity between the three treatment groups at the 12-week follow-up. Depending on which model (Model 1 or 2) was used, there were three to four fixed effects and the participants were taken as random effects. This was completed to determine the effect of treatment group allocation, pain phenotype, baseline pain intensity and the number of physiotherapy visits on pain intensity at 12-week follow-up.

Linear mixed model 1: Controlling for treatment group allocation, pain intensity at baseline and number of physiotherapy visits.

In the linear mixed model 1, and in reference to treatment group allocation, pedometer-based walking (W) was the reference group. Treatment group allocation was not a statistically significant predictor

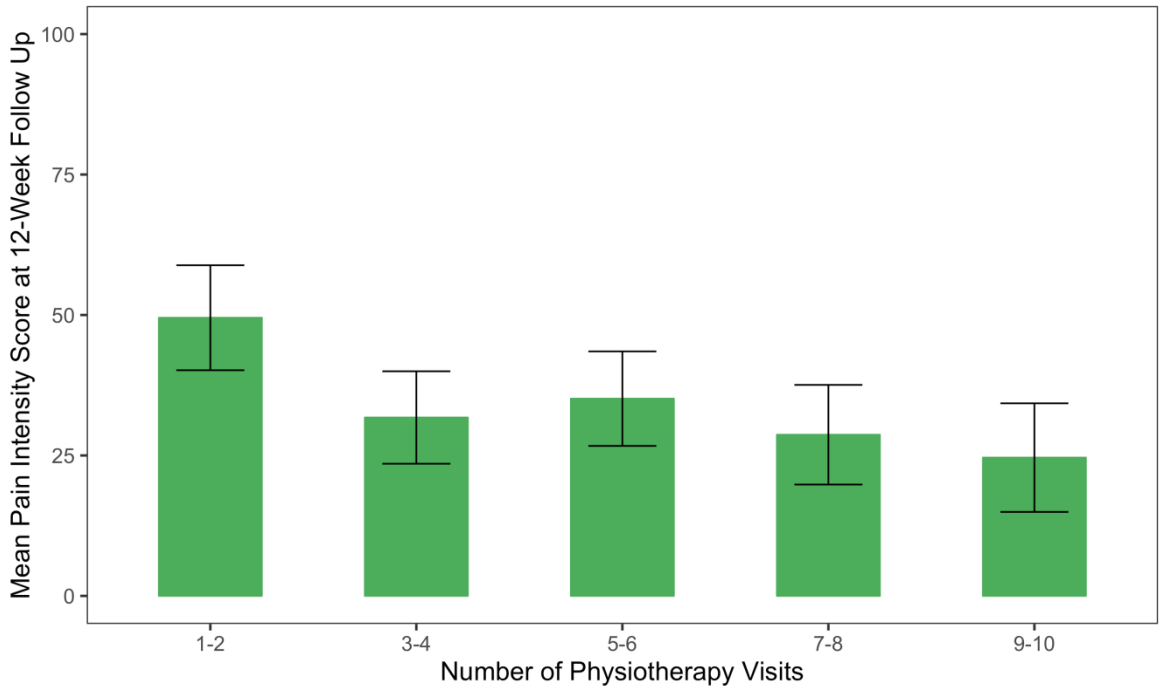
of pain intensity between treatment groups at 12-week follow-up (Table 42). After controlling for all other variables in the model (treatment by group and number of physiotherapy visits), pain intensity at baseline was a statistically significant predictor of pain intensity at 12-week follow-up ($p<0.01$). A one-unit increment in baseline pain intensity score being associated with a 0.5 unit increase in pain intensity at 12-week follow-up (Table 42). After controlling for the other variables in the model (treatment by group and pain at baseline), the number of physiotherapy visits was also a statistically significant predictor of pain intensity at 12-week follow-up ($p=0.01$) (Table 42). Those who attended the physiotherapist on one – two occasions were most likely to have no significant change in pain intensity at 12-week follow-up. Post-hoc tests completed only on physiotherapy visits showed that the pain intensity score at 12-week follow-up was statistically significantly greater for participants who had attended one to two physiotherapy visits than that for three to four, seven to eight and nine to ten physiotherapy visits ($p=0.03$, 0.02 and 0.01 respectively) (Figure 12).

Table 42: Linear mixed model 1- fixed effects output exploring contributions of treatment group allocation, pain intensity at baseline, and number of physiotherapy visits to pain intensity at 12-week follow-up.

Effect	Estimates	Confidence intervals	Df	T	p value
Intercept	-1.34	-16.82 to 14.13	145	-0.17	0.9
Treatment PW	-7.33	-17.66 to 3.00	2	-1.39	0.17
Treatment P	-0.53	-10.72 to 9.66	2	-0.01	0.92
Treatment W. Reference group	0		2		
Pain intensity at baseline	0.53	0.35 to 0.71	1	5.85	<0.01***
Number of physio visits 1-2	24.89	11.13 to 38.66	4	3.54	0.01***
Number of physio visits 3-4	7.13	-5.74 to 20.01	4	1.09	0.28
Number of physio visits 5-6	10.49	-2.11 to 23.09	4	1.63	0.10
Number of physio visits 7-8	4.07	-8.62 to 16.77	4	0.63	0.53
Number of physio visits 9-10. Reference group	0		4		

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Figure 12: Mean pain intensity scores against the number of physiotherapy visits throughout the intervention period.



Linear mixed model 2: Controlling for treatment group allocation, pain phenotype, number of physiotherapy visits and interaction of treatment group allocation and pain phenotype.

Model 2 was constructed to exclude pain intensity at baseline as it was a confounding variable when used with pain phenotype in the analysis. In reference to treatment group allocation, W treatment group was the reference group. Treatment group allocation was not a statistically significant predictor of pain intensity between treatment groups at 12-week follow-up (Table 43).

Table 43: Linear mixed model 2 - fixed effects output exploring contributions of treatment group allocation, pain phenotype, and number of physiotherapy visits, and interactions between treatment group allocation and pain phenotype to pain intensity at 12-week follow-up.

Effect	Estimates	Confidence intervals	df	t	p value
Intercept	23.04	8.96 to 37.12	145	3.21	0.00
Treatment PW	-10.88	-25.26 to 3.49	2	-1.48	0.14
Treatment P	-1.14	-15.40 to13.13	2	-0.16	0.88
Treatment W. Reference group	0		2		
Neuropathic pain phenotype	14.91	1.05 to 28.76	1	2.11	0.04*
Nociceptive pain phenotype. Reference group	0		1		
Number of physio visits 1-2	20.71	6.08 to 35.33	4	2.78	0.01**
Number of physio visits 3-4	6.32	-7.33 to 19.96	4	0.91	0.37
Number of physio visits 5-6	6.07	-7.21 to 19.35	4	0.9	0.37
Number of physio visits 7-8	0.65	-12.91 to 14.21	4	0.09	0.93
Number of physio visits 9-10. Reference group	0		4		
Treatment PW x Neuropathic Pain phenotype	8.04	11.77 to 27.85	2	0.8	0.43
Treatment PW x Nociceptive Pain phenotype. Reference group	0		2		
Treatment P x Neuropathic Pain phenotype	-1.99	-21.99 to 17.99	2	-0.2	0.84
Treatment P x Nociceptive Pain phenotype. Reference group	0		2		
Treatment W x Neuropathic Pain phenotype. Reference group	0		2		
Treatment W x Nociceptive Pain phenotype. Reference group	0		2		

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

After controlling for all other variables in the model (treatment by group, number of physiotherapy visits, and the interaction of treatment by group and pain phenotype), pain phenotype was a statistically significant predictor of pain intensity at 12-week follow-up (p=0.04) (Table 43). Neuropathic pain phenotype predicted greater pain intensity at 12-week follow up.

After controlling for the other variables in the model (pain phenotype, treatment by group, and the interaction of treatment by group and pain phenotype), the effect of the number of physiotherapy visits on pain intensity score at the 12-week follow-up demonstrated there was a statistically significant association with the number of physiotherapy visits. One - two physiotherapy visits associated with greater pain intensity at 12-week follow-up (p=0.01) (Table 43). This finding is the same as in Model 1.

Investigate analysis

A linear mixed model regression analysis was conducted to determine, whether any of the independent variables (Table 41) were associated with pain intensity at 12-week follow-up, thereby investigating factors predicting pain intensity in all participants at 12-week follow-up. The models (3 and 4) included independent variables typically associated with CLBP (Table 41). Pain intensity at baseline, treatment group allocation, time point at six-week follow-up, time point at 12-week follow-up, number of physiotherapy visits, and pain duration were also included.

Linear mixed model 3: Looking for changes in pain intensity controlling for all independent variables except pain phenotype.

The model 3 showed both pain intensity at baseline ($p<0.01$) and number of physiotherapy visits ($p=0.01$) remained statistically significant predictors of the pain intensity outcome at 12-week follow-up (Table 44).

Table 44: Linear mixed model 3- fixed effects output exploring contributions of variables to pain intensity at 12-week follow-up.

Effect	Estimates	Confidence intervals	df	t	p value
Intercept	11.5	-19.51 to 42.51	133	0.73	0.46
Treatment PW.	-9.95	-20.42 to 0.51	140	-1.86	0.06
Treatment P.	-2.85	-13.19 to 7.49	137	-0.54	0.59
Treatment W. Reference group	0				
Pain intensity at baseline	0.55	0.38 to 0.71	130	6.66	<0.01***
Number of physio visits 1-2	16.78	3.99 to 29.58	130	2.57	0.01*
Number of physio visits 3-4	1.24	-10.33 to 12.81	130	0.21	0.83
Number of physio visits 5-6	6.84	-4.71 to 18.39	130	1.16	0.24
Number of physio visits 7-8	-0.70	-12.18 to 10.77	130	-0.12	0.90
Number of physio visits 9-10. Reference group	0				
Six-week follow-up	1.12	-4.99 to 7.22	117	0.36	0.72
12-week follow-up. Reference group	0				
Age	0.01	-0.34 to 0.35	130	0.03	0.97
Gender female	2.30	0.54 to 9.59	3.7145	130	0.62
Gender male. Reference group	0				
Employed	0.96	-11.83 to 13.74	130	0.15	0.88
Unemployed. Reference group	0				
Education: university/post grad.	-1.1734	-9.97 to 7.62	130	-0.26	0.79
Education: Diploma	2.6899	-5.62 to 11.00	130	0.63	0.52
Education: Completed school. Reference group	0				
Smoker: no	-2.27	-9.58 to 5.05	130	-0.61	0.54
Smoker: yes. Reference group	0				
BMI	-0.35	-1.03 to 0.33	130	-1.02	0.31
Pain Duration	0.14	-0.30 to 0.59	130	0.63	0.53
Treatment PW x Six-week follow-up.	4.17	-4.65 to 12.98	117	0.93	0.35
Treatment PW x 12-week follow-up. Reference group	0				
Treatment P x Six-week follow-up	-3.75	-12.66 to 5.17	117	-0.82	0.41
Treatment P x 12-week follow-up. Reference group	0				
Treatment W x Six-week follow-up. Reference group	0				
Treatment W x 12-week follow-up. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

After controlling for all other variables in the model including treatment by group, number of physiotherapy visits, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, interaction of treatment by group and six-week follow-up, and interaction of treatment by group and 12-week follow-up, pain intensity at base-line was a statistically significant predictor of pain intensity at 12-week follow-up ($p<0.01$). A one-unit increment in baseline pain intensity score being associated with a 0.5-unit increase in pain intensity at 12-week follow-up (Table 44).

After controlling for the other variables in the model including treatment by group, pain intensity at baseline, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, interaction of treatment by group and six-week follow-up, and interaction of treatment by group and 12-week follow-up, the effect of the number of physiotherapy visits on pain intensity score at 12-week follow-up also demonstrated a statistically significant association with the number of physiotherapy visits ($p=0.01$). Fewer physiotherapy visits were associated with greater pain intensity at 12-week follow-up (Table 44).

All of the other predictors were not statistically significantly associated with pain intensity at 12-week follow-up in this model (treatment by group, six-week follow-up, 12-week follow-up, age, gender, employment, education, smoking status, BMI, and pain duration).

Linear mixed model 4: Looking for changes in pain intensity controlling for all independent variables except baseline pain intensity.

Model 4 was constructed to exclude pain intensity at baseline as it was a confounding variable when used with pain phenotype in the analysis. Treatment group allocation was not a statistically significant predictor of pain intensity at 12-week follow-up (Table 45). After controlling for other variables in the model including treatment by group, number of physiotherapy visits, week (six-week follow-up, 12-week follow-up), age, gender, employment status, education status, smoking, BMI, pain duration, interaction of treatment by group and pain phenotype, interaction of treatment by group and six-week follow-up, interaction of treatment by group and 12-week follow-up, interaction of pain phenotype and six-week follow-up, interaction of pain phenotype and 12-week follow-up, neuropathic pain phenotype was shown to be a statistically significant predictor of greater pain intensity at 12-week follow-up ($p=0.01$) (Table 45). The effects of the other variables in the model were not statistically significant.

Table 45: Linear mixed model 4- fixed effects output exploring contributions of variables to pain intensity at 12-week follow-up.

Effect	Estimates	Confidence intervals	df	t	p value
Intercept	35.93	3.06 to 68.80	133	2.14	0.03
Treatment PW	-5.49	-19.70 to 8.71	150	-0.76	0.45
Treatment P	-1.00	-15.50 to 13.49	150	-0.14	0.89
Treatment W. Reference group	0				
Neuropathic pain phenotype	21.05	7.06 to 35.04	149	2.95	0.01**
Nociceptive pain phenotype. Reference group	0		.	.	.
Number of physio visits 1-2	11.39	-2.76 to 25.56	128	1.58	0.12
Number of physio visits 3-4	-1.18	-13.96 to 11.59	128	-0.18	0.86
Number of physio visits 5-6	1.16	-11.54 to 13.87	128	0.18	0.86
Number of physio visits 7-8	-2.87	-15.55 to 9.81	128	-0.44	0.66
Number of physio visits 9-10. Reference group	0		.	.	.
Six-week follow-up	0.46	-6.51 to 7.44	115	0.13	0.89
12-week follow-up. Reference group	0				
Age	-0.12	-0.50 to 0.26	128	-0.62	0.54
Gender female	6.00	-2.17 to 14.18	128	1.44	0.15
Gender male. Reference group	0				
Employed	-3.12	-17.27 to 11.02	128	-0.43	0.66
Unemployed. Reference group	0				
Education: university/post grad.	0.67	-9.24 to 10.58	128	0.13	0.89
Education: Diploma	0.74	-8.47 to 9.95	128	0.16	0.87
Education: Completed school. Reference group	0		.	.	.
Smoker: no	-1.99	-10.23 to 6.23	128	-0.48	0.63
Smoker: yes. Reference group	0				
BMI	-0.24	-0.99 to 0.51	128	-0.63	0.53
Pain Duration	0.13	-0.37 to 0.64	128	0.52	0.60
Treatment PW x Neuropathic Pain phenotype	-6.25	-24.31 to 11.80	128	-0.68	0.49
Treatment PW x Nociceptive Pain phenotype. Reference group	0				
Treatment P x Neuropathic Pain phenotype	-9.50	-28.52 to 9.51	128	-0.98	0.32
Treatment P x Nociceptive Pain phenotype. Reference group	0				
Treatment W x Neuropathic Pain phenotype. Reference group	0				
Treatment W x Nociceptive Pain phenotype. Reference group	0				

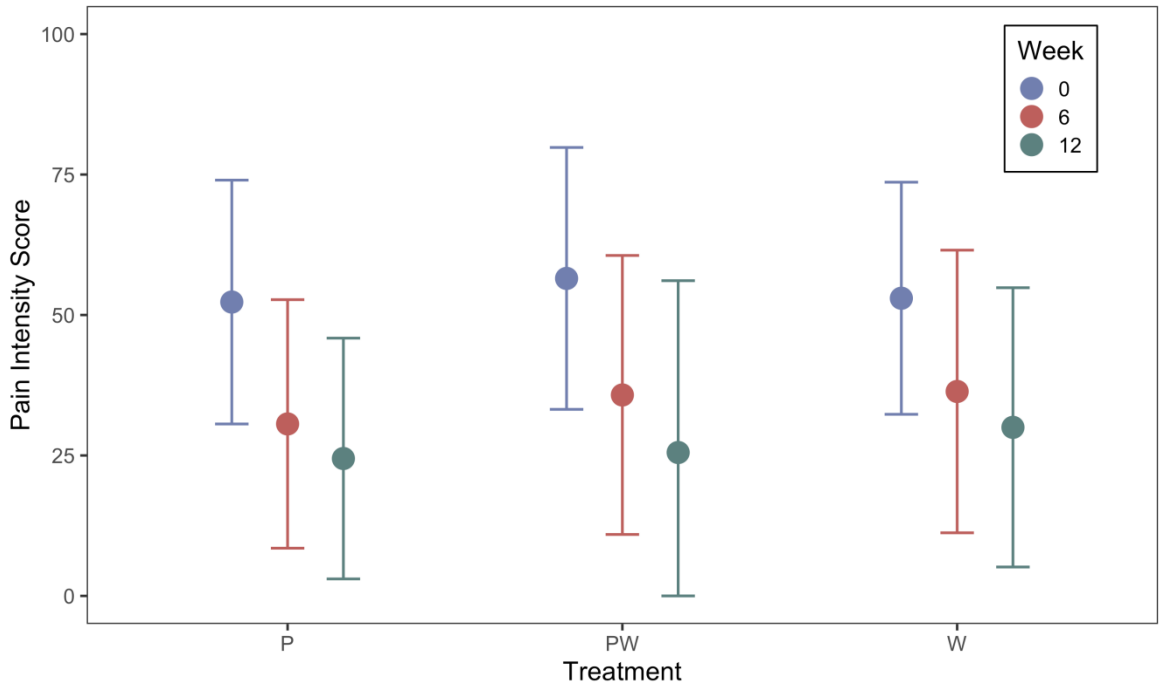
Effect	Estimates	Confidence intervals	df	t	p value
Treatment PW x Six-week follow-up	3.11	-5.84 to 12.06	115	0.68	0.49
Treatment PW x 12-week follow-up. Reference group	0				
Treatment P x Six-week follow-up	-3.86	-12.96 to 5.23	115	0.83	0.40
Treatment P x 12-week follow-up. Reference group	0				
Treatment W x Six-week follow-up. Reference group	0				
Treatment W x 12-week follow-up. Reference group	0				
Neuropathic pain phenotype x Six-week follow-up	1.53	-5.89 to 8.97	115	0.41	0.68
Neuropathic pain phenotype x 12-week follow-up. Reference group	0				
Nociceptive pain phenotype x Six-week follow-up. Reference group	0				
Nociceptive pain phenotype x Six-week follow-up. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

To summarise the primary outcome of pain intensity, information for change in pain intensity from baseline to 12-week follow-up: the primary analysis (ANOVA and linear mixed model 1 and 2), together with further investigative analysis using linear mixed models (models 3 and 4) including all the predictors in Table 41, concluded treatment group allocation was not significant predictor of reduction in pain intensity at 12-week follow-up. However, both linear mixed models in the primary and investigative analyses showed the number of physiotherapy visits did predict a reduction in pain intensity at 12-week follow-up. Both primary and investigative analyses show that baseline pain intensity and pain phenotype were statistically significant predictors of a decrease in pain intensity at 12-week follow-up.

Figure 13 shows changes in pain intensity between baseline and follow-up at six-weeks and 12-weeks within treatment groups. The linear mixed model regression analyses showed there was no statistically significant effect of time (follow-up at six-weeks and 12-weeks), treatment group allocation, or interaction between treatment and time.

Figure 13: Comparison of pain intensity within each treatment group over time.



P, usual care treatment; **W,** pedometer-based walking intervention;

PW, usual care treatment and pedometer-based walking intervention;

Week 0, baseline; **Week 6**, six-week follow-up; **Week 12**, 12-week follow-up

Secondary outcome measures analyses

Secondary outcomes measured in this RCT included, disability, kinesiophobia and pain catastrophizing.

A secondary outcome measure analysis was completed using an ANOVA, individually comparing the change in secondary outcomes (ODI, TSK and PCS) from baseline to 12-week follow-up, between treatment groups. In addition, for every individual secondary outcome measure, linear mixed models were constructed. Using the linear mixed models, analyses were conducted using covariates (Table 41) to examine if there were changes in any of the secondary outcome measures, and what the predictors were of these outcome measures at 12-week follow-up.

Disability

The Oswestry Disability Index (ODI) was used to measure disability related to CLBP. Participants baseline ODI mean score for the entire cohort was 24.9 (SD 11.2) (Table 37).

An analysis for ODI investigated whether there was a significant difference in ODI score between treatment groups at 12-week follow-up. An ANOVA showed no statistically significant difference

in the change in ODI score from baseline to 12-week follow-up between groups at 12-week follow-up ($F(2,104) = 0.42$, $p = 0.66$).

Linear mixed models 5 and 6 were generated for a comparison of ODI score between the three treatment groups at 12-week follow-up. Due to baseline ODI score and pain phenotype being confounding variables when used together in a single model, results were presented in models 5 and 6. Model 5 controlled for baseline ODI score and excluded pain phenotype. Model 6 controlled for pain phenotype and excluded baseline ODI score. However, depending on which model was used (model 5 or 6), the analysis was used for determination of the effect of treatment group allocation, pain phenotype, baseline disability, and selected independent variables (Table 41) on the ODI score at 12-week follow-up. Associated variables that are commonly associated with pain intensity were likewise included in the analysis of ODI at 12-week follow-up. These included: week (six-week follow-up and 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, and pain intensity at 12-week follow-up. These variables were taken as the fixed effects and the participants were taken as random effects.

Linear mixed model 5: Looking for changes in ODI score controlling for all independent variables except baseline pain phenotype and number of physiotherapy visits.

Controlling for variables, including ODI score at baseline, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, pain intensity at 12-week follow-up, and interaction of treatment group allocation and six-week follow-up and interaction of treatment group allocation and 12-week follow-up on ODI score at 12-week follow-up, there was no statistically significant effect of treatment group allocation on ODI score at 12-week follow-up ($p=0.23$ for PW and $p=0.86$ for P) (Table 46). However, controlling for variables described in table 41, ODI score at baseline ($p<0.01$), and at six-week follow-up ($p=0.02$), along with age ($p=0.05$) and pain intensity at 12-week follow-up ($p<0.01$) predicted ODI score at 12-week follow-up (Table 46). One-unit increment in ODI scores at baseline being associated with a 0.5 unit increase in ODI score at 12-week follow-up. The ODI mean score was higher at six-week follow-up compared to 12-week follow-up. Additionally, the model indicted that there was a 0.1 unit increase in ODI score for every one-year increase in age. Furthermore, for every 1-unit increase in pain intensity score at 12-week follow-up the ODI score increased by 0.2 units at 12-week follow-up. No other statistically significant predictors of ODI score at 12-week follow-up were found.

Table 46: Linear mixed model 5- fixed effects output exploring contributions of variables to ODI score at 12-week follow-up.

Effect	Estimates	Confidence intervals	df	t	p value
Intercept	-8.47	-15.40 to -1.53	147	-2.39	0.02
Treatment PW	-1.63	-4.30 to 1.04	137	-1.2	0.23
Treatment P	0.23	-2.49 to 2.96	138	0.17	0.86
Treatment W. Reference group	0		.	.	.
ODI at baseline	0.51	0.42 to 0.60	147	11.25	<0.01***
Six-week follow-up	2.57	0.49 to 4.64	124	2.43	0.02*
12-week follow-up. Reference group	0				
Age	0.09	0.00 to 0.18	137	1.99	0.05*
Gender female	-0.82	-2.73 to 1.08	136	-0.85	0.39
Gender male. Reference group	0		.	.	.
Employed	1.63	-1.80 to 5.07	136	0.93	0.35
Unemployed. Reference group	0		.	.	.
Education: university/post grad.	0.27	-2.01 to 2.57	137	0.24	0.81
Education: Diploma	0.67	-1.51 to 2.87	136	0.61	0.54
Education: Completed school. Reference group	0		.	.	.
Smoker: no	0.76	-1.16 to 2.69	137	0.78	0.43
Smoker: yes. Reference group	0		.	.	.
BMI	0.02	-0.15 to 0.20	136	0.24	0.81
Pain Duration	0.03	-0.09 to 0.15	136	0.47	0.63
Pain intensity at 12-week follow-up	0.24	0.20 to 0.27	240	13.89	<0.01***
Treatment PW x Six-week follow-up	-0.27	-3.24 to 2.70	125	-0.18	0.85
Treatment PW x 12-week follow-up. Reference group	0		.	.	.
Treatment P x Six-week follow-up	-0.23	-3.26 to 2.79	125	-0.15	0.87
Treatment P x 12-week follow-up. Reference group	0				
Treatment W x Six-week follow-up. Reference group	0				
Treatment W x 12-week follow-up. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Linear mixed model 6: Looking for changes in ODI score controlling for all independent variables except baseline ODI score and number of physiotherapy visits.

Controlling for variables including, pain phenotype, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, pain intensity at 12-week follow-up, interaction of treatment group allocation and pain phenotype, interaction of treatment

group allocation and six-week follow-up, interaction of treatment group allocation and 12-week follow-up, interaction of pain phenotype and 6-week follow-up, and interaction of pain phenotype and 12-week follow-up described in Table 47, there was no statistically significant effect of treatment group allocation on ODI score at 12-week follow-up (p=0.56 for PW and p=0.59 for P). However, controlling for variables described in Table 47, pain phenotype (p=0.01), along with employment status (p=0.03) and pain intensity at 12-week follow-up (p<0.01) predicted ODI score at 12-week follow-up. Having a neuropathic pain phenotype was predictive of a greater ODI score at 12-week follow-up. Being unemployed was a statistically significant predictor of higher ODI scores at 12-week follow-up. Furthermore, for every 1-unit increase in pain intensity score at 12-week follow-up the ODI score increased by 0.3 units at 12-week follow-up. No other statistically significant predictors of ODI score at 12-week follow-up were found.

Table 47: Linear mixed model 6 - fixed effects output exploring contributions of variables to ODI score at 12-week follow-up.

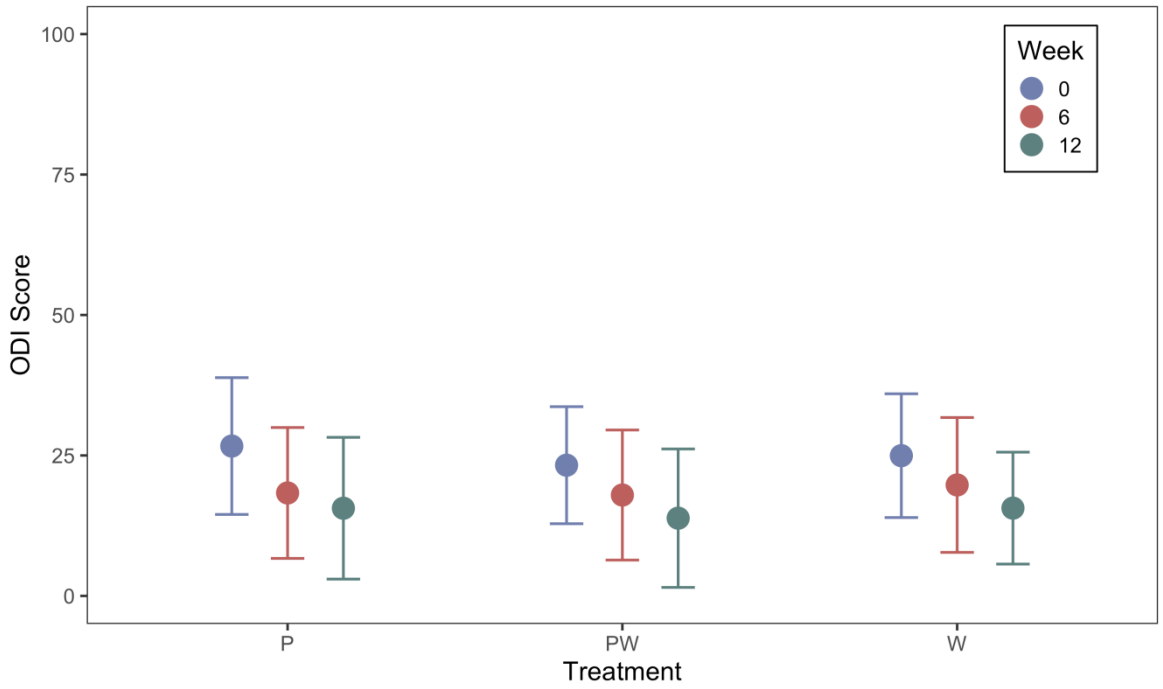
Effect	Estimates	Confidence intervals	df	t	p value
Intercept	-3.50	-12.01 to 5.00	145	-0.81	0.42
Treatment PW	-1.14	-5.08 to 2.78	156	-0.57	0.56
Treatment P	1.08	-2.96 to 5.14	155	0.53	0.59
Treatment W. Reference group	0				
Neuropathic pain phenotype	6.30	2.15 to 10.45	156	2.98	0.01**
Nociceptive pain phenotype. Reference group	0				
Six-week follow-up	1.98	-0.42 to 4.39	110	1.62	0.10
12-week follow-up. Reference group	0				
Age	0.10	-0.01 to 0.21	134	1.81	0.07
Gender female	-0.57	-2.91 to 1.75	134	-0.49	0.62
Gender male. Reference group	0		.	.	.
Unemployed	4.41	0.34 to 8.48	134	2.13	0.03*
Employed: yes. Reference group	0		.	.	.
Education: university/post grad.	2.10	-0.68 to 4.90	134	1.48	0.14
Education: Diploma	1.78	-0.84 to 4.40	134	1.33	0.18
Education: Completed school. Reference group	0		.	.	.
Smoker: no	0.48	-1.85 to 2.82	134	0.41	0.68
Smoker: yes. Reference group	0		.	.	.
BMI	0.06	-0.14 to 0.28	134	0.63	0.52
Pain Duration	0.13	-0.01 to 0.27	134	1.8	0.07
Pain intensity at 12-week follow-up	0.27	0.23 to 0.31	247	14.14	<0.01***
Treatment PW x Neuropathic Pain phenotype	-3.21	-8.47 to 2.03	134	-1.2	0.23
Treatment PW x Nociceptive Pain phenotype. Reference group	0		.	.	.

Effect	Estimates	Confidence intervals	df	t	p value
Treatment P x Neuropathic Pain phenotype	-1.63	-7.13 to 3.87	134	-0.58	0.56
Treatment P x Nociceptive Pain phenotype. Reference group	0		.	.	.
Treatment W x Neuropathic Pain phenotype. Reference group	0		.	.	.
Treatment W x Nociceptive Pain phenotype. Reference group	0		.	.	.
Treatment PW x Six-week follow-up	-0.49	-3.58 to 2.60	111	-0.31	0.75
Treatment PW x 12-week follow-up. Reference group	0				
Treatment P x Six-week follow-up	-0.22	-3.38 to 2.93	111	-0.14	0.88
Treatment P x 12-week follow-up. Reference group	0		.	.	.
Treatment W x Six-week follow-up. Reference group	0		.	.	.
Treatment W x12-week follow-up. Reference group	0		.	.	.
Neuropathic pain phenotype x Six-week follow-up	1.29	-1.27 to 3.87	110	0.99	0.32
Neuropathic pain phenotype x 12-week follow-up. Reference group	0		.	.	.
Nociceptive pain phenotype x Six-week follow-up. Reference group	0		.	.	.
Nociceptive pain phenotype x 12-week follow-up. Reference group	0		.	.	.

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Figure 14 shows changes in ODI score within treatment groups between baseline and follow-up at six-weeks and 12-weeks. All groups followed a trend in decreasing disability score between baseline and follow-up at 12-weeks.

Figure 14: Comparison of ODI score within each treatment group over time.



P, usual care treatment; **W,** pedometer-based walking intervention;
PW, usual care treatment and pedometer-based walking intervention;
Week 0, baseline; **Week 6**, six-week follow-up; **Week 12**, 12-week follow-up

Kinesiophobia

The Tampa Scale of Kinesiophobia (TSK) was used to measure fear of movement related to CLBP. The mean score for kinesiophobia on the TSK for the entire cohort at baseline was 38/68 indicative of a fear avoidance behaviour, since the cut off score was 37/68 with scores of 37 and above categorized as kinesiophobic (Table 37).

An ANOVA was used to look for changes in kinesiophobia, between treatment groups between baseline and follow-up at 12-week follow-up. There was no statistically significant difference in TSK mean scores between baseline and 12-week follow-up between treatment groups at 12-week follow-up ($F(2,104) = 1.49$, $p = 0.23$).

There was no association with baseline score of TSK and pain phenotype, therefore only one model was generated. Linear mixed model 7 was generated for a comparison of TSK score between the three treatment groups at 12-week follow-up. The analysis was used to determine the effect of treatment group allocation, pain phenotype, baseline TSK score, number of physiotherapy visits, and selected independent variables (Table 41) on the TSK score at 12-week follow-up. Associated variables that are commonly associated with pain intensity were included in the analysis of TSK at 12-week follow-up. These included: week (six-week follow-up and 12-week follow-up), age, gender,

employment, education, smoking status, BMI, and pain duration. Pain intensity at 12-week follow-up was excluded from the analysis because it was associated with number of physiotherapy visits. These variables were taken as the fixed effects and the participants were taken as random effects.

Linear mixed model 7: Looking for changes in TSK score controlling for all independent variables except pain intensity at 12-week follow-up.

After controlling for variables including TSK score at baseline, pain phenotype, number of physiotherapy visits, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, interaction of treatment by group and pain phenotype, interaction of treatment group allocation and six-week follow-up, interaction of treatment group allocation and 12-week follow-up, interaction of pain phenotype and six-week follow-up, and interaction of pain phenotype and 12-week follow-up described in Table 48, there was no statistically significant effect of treatment group allocation on TSK score at 12-week follow-up (p=0.34 for PW and p=0.47 for P).

However, after controlling for variables in the model described in Table 48, TSK score at baseline (p<0.01), and number of physiotherapy visits (p=0.01) predicted TSK score at 12-week follow-up. A one-unit increment in baseline TSK scores being associated with a 0.8 unit increase in TSK score at 12-week follow-up. The effect of the number of physiotherapy visits on TSK score at the 12-week follow-up demonstrated there was a statistically significant association with the number of physiotherapy visits. One - two physiotherapy visits were associated with greater TSK scores. No other statistically significant predictors of TSK score at 12-week follow-up were found.

Table 48: Linear mixed model 7 - fixed effects output exploring contributions of variables to TSK score at 12-week follow-up.

Effect	Estimates	Confidence intervals	df	t	p value
Intercept	1.85	-7.23 to 10.94	129	0.4	0.69
Treatment PW	-2.86	-6.06 to 0.34	145	-1.75	0.08
Treatment P	1.18	-2.09 to 4.47	145	0.71	0.47
Treatment W. Reference group	0				
TSK Baseline	0.75	0.61 to 0.87	126	11.06	<0.01***
Neuropathic pain phenotype	1.56	-1.63 to 4.77	145	0.96	0.33
Nociceptive pain phenotype. Reference group					
Physiotherapy visits 1-2	3.84	0.65 to -7.03	126	2.36	0.01*
Physiotherapy visits 3-4	-0.74	-3.66 to 2.16	126	-0.5	0.61
Physiotherapy visits 5-6	-0.45	-3.31 to 2.40	126	-0.31	0.75

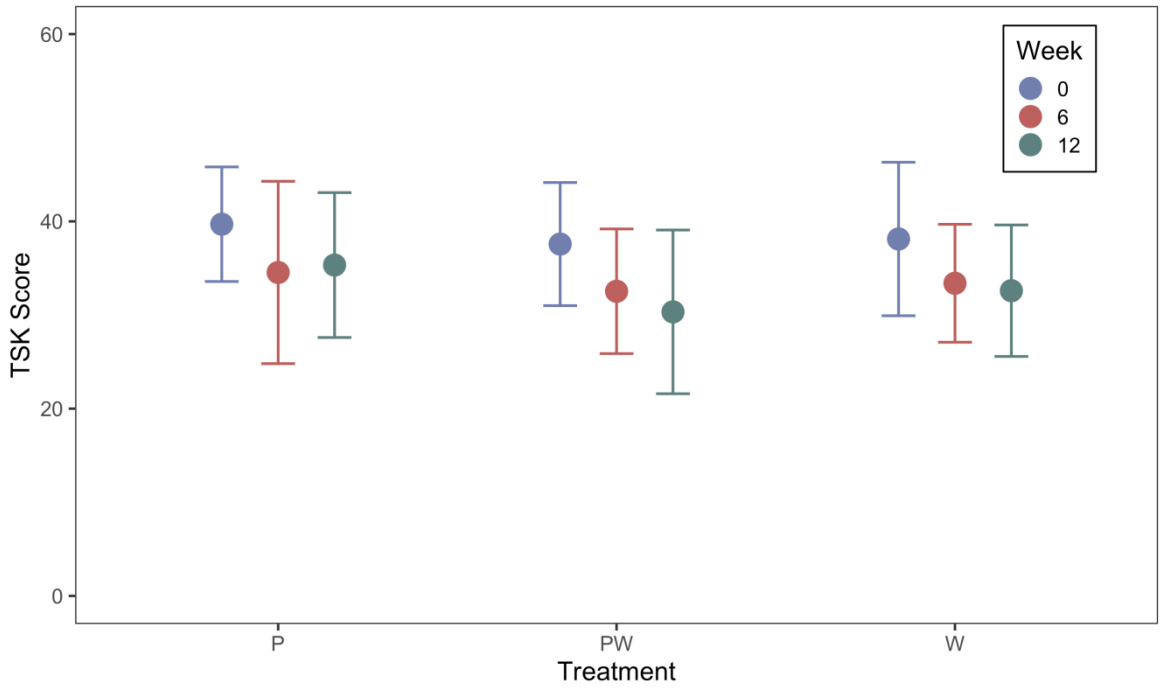
Effect	Estimates	Confidence intervals	df	t	p value
Physiotherapy visits 7-8	-0.52	-3.36 to 2.31	126	-0.36	0.71
Physiotherapy visits 9-10. Reference group	0				
Six-week follow-up	0.87	-1.04 to 2.79	111	0.89	0.37
12-week follow-up. Reference group	0				
Age	-0.00	-0.08 to 0.08	126	-0.06	0.94
Gender female	1.05	-0.79 to 2.90	126	1.11	0.26
Gender male. Reference group	0				
Unemployed	-0.99	-4.21 to 2.23	126	-0.6	0.54
Employed. Reference group	0				
Education: university/post grad.	1.71	-0.50 to 3.93	126	1.51	0.13
Education: Diploma	0.42	-1.65 to 2.51	126	0.40	0.68
Education: Completed school. Reference group	0				
Smoker: no	1.72	-0.12 to 3.57	126	1.83	0.06
Smoker: yes. Reference group	0				
BMI	0.07	-0.09 to 0.25	126	0.89	0.37
Pain Duration	-0.09	-0.21 to 0.01	126	-1.7	0.09
Treatment PW x Neuropathic Pain phenotype	1.06	-2.98 to 5.11	126	0.52	0.60
Treatment PW x Nociceptive Pain phenotype. Reference group	0				
Treatment P x Neuropathic Pain phenotype	-0.10	-4.39 to 4.17	126	-0.05	0.96
Treatment P x Nociceptive Pain phenotype. Reference group	0				
Treatment W x Neuropathic Pain phenotype. Reference group	0				
Treatment W x Nociceptive Pain phenotype. Reference group	0				
Treatment PW x Six-week follow-up	0.79	-1.64 to 3.24	111	0.64	0.52
Treatment PW x 12-week follow-up. Reference group	0				
Treatment P x six-week follow-up	-0.63	-3.13 to 1.85	111	-0.5	0.61
Treatment P x 12-week follow-up. Reference group	0				
Treatment W x Six-week follow-up. Reference group	0				
Treatment W x 12-week follow-up. Reference group	0				

Effect	Estimates	Confidence intervals	df	t	p value
Neuropathic pain phenotype x Six-week follow-up	0.43	-2.47 to 1.59	111	-0.42	0.67
Neuropathic pain phenotype x 12-week follow-up. Reference group	0				
Nociceptive pain phenotype x Six-week follow-up. Reference group	0				
Nociceptive pain phenotype x 12-week follow-up. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Figure 15 demonstrates the changes in TSK score within treatment groups between baseline and follow-up at six-weeks and 12-weeks. The linear mixed model regression analyses showed there was no statistically significant effect of time (six-weeks and 12-weeks follow-up), treatment group allocation, or interaction between treatment group allocation and time.

Figure 15: Comparison of TSK score within each treatment group over time.



P, usual care treatment; **W,** pedometer-based walking intervention;

PW, usual care treatment and pedometer-based walking intervention;

Week 0, baseline; **Week 6**, six-week follow-up; **Week 12**, 12-week follow-up

Pain catastrophizing

The Pain Catastrophizing Scale (PCS) was used to measure pain catastrophizing related to CLBP. The mean score for pain catastrophizing on the PCS for the entire cohort at baseline was 18/52

(Table 37). This indicates low levels of pain catastrophizing in this cohort, with scores greater than 30/52 indicating high levels of catastrophizing.

An ANOVA was used to determine any changes in PCS score between treatment groups, between baseline and follow-up at 12-week follow-up. There was no statistically significant difference in PCS scores between baseline and 12-week follow-up between treatment groups at 12-week follow-up $(2,104) = 0.32$, $p = 0.73$).

There was no association with baseline score of PCS and pain phenotype, therefore only one model was generated. Linear mixed model 8 was generated for a comparison of PCS score between the three treatment groups at 12-week follow-up. The analysis was used for determination of the effect of treatment group allocation, pain phenotype, baseline PCS score, number of physiotherapy visits, and selected independent variables (Table 41) on the PCS score at 12-week follow-up. Associated variables that are commonly associated with pain intensity were likewise included in the analysis of PCS at 12-week follow-up. These included: week (six-week follow-up and 12-week follow-up), age, gender, employment, education, smoking status, BMI, and pain duration. Pain intensity at 12-week follow-up was excluded from the analysis because it was associated with number of physiotherapy visits. These variables were taken as the fixed effects and the participants were taken as random effects.

Linear mixed model 8: Looking for changes in PCS score controlling for all independent variables except pain intensity at 12-week follow-up.

After controlling for variables including treatment group allocation, pain phenotype, number of physiotherapy visits, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, interaction of treatment group allocation and pain phenotype, interaction of treatment group allocation and week, and interaction of pain phenotype and week, PCS score at baseline was found to be a statistically significant predictor of PCS at 12-week follow-up ($p<0.01$) (Table 49). For every 1-unit increase in the PCS baseline score, the PCS score at 12-week follow-up increased by 0.6 units. After controlling for treatment group allocation, PCS score at baseline, pain phenotype, week (six-week follow-up, 12-week follow-up), age, gender, employment, education, smoking status, BMI, pain duration, interaction of treatment group allocation and pain phenotype, interaction of treatment group allocation and week, and interaction of pain phenotype and week, the number of physiotherapy visits was found to be a predictor of PCS score at 12-week follow-up. Attending 1-2 physiotherapy visits was shown to be a predictor of a greater PCS score at 12-week follow-up ($p=0.01$) (Table 49).

Table 49: Linear mixed model 8 - fixed effects output exploring contributions of variables to PCS score at 12-week follow-up.

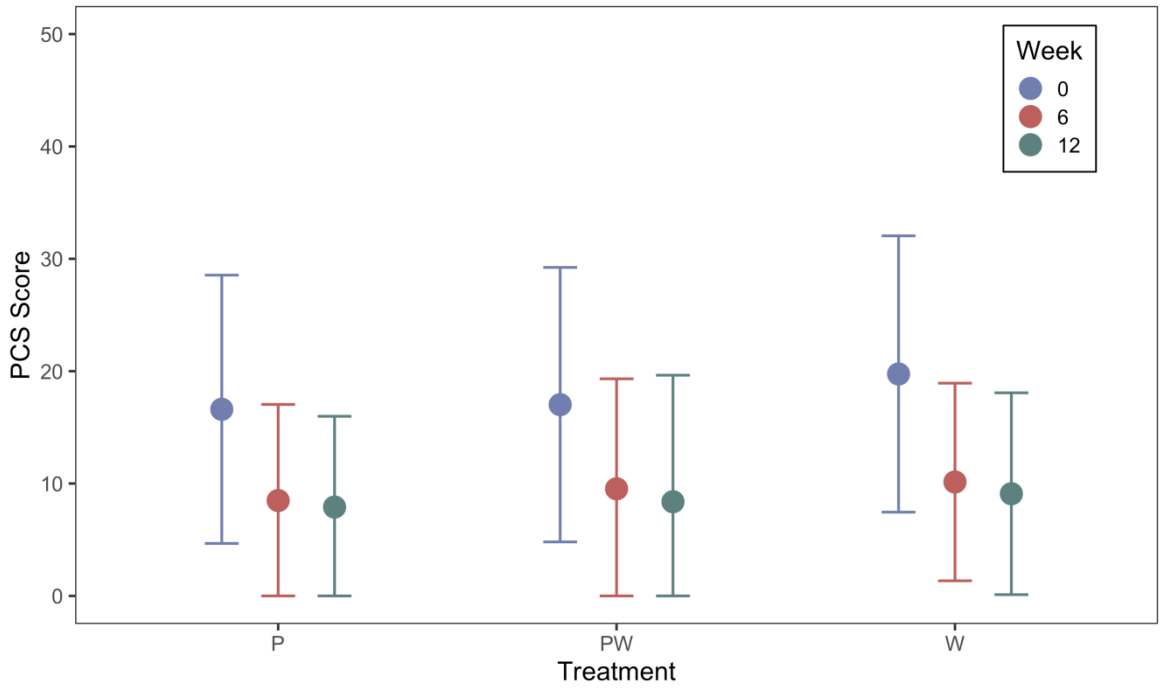
Effect	Estimates	Confidence intervals	df	t	p value
Intercept	-2.93	-14.03 to 8.15	131	-0.52	0.60
Treatment PW	-2.77	-7.37 to 1.82	142	-1.18	0.23
Treatment P	0.90	-3.80 to 5.61	142	0.38	0.70
Treatment W. Reference group	0				
Pain catastrophizing at baseline	0.59	0.47 to 0.71	128	9.94	<0.01***
Neuropathic pain phenotype	1.57	-3.10 to 6.25	141	0.66	0.50
Nociceptive pain phenotype. Reference group	0				
Number of physio visits 1-2	7.89	3.21 to 12.56	128	3.31	0.01**
Number of physio visits 3-4	-0.40	-4.64 to 3.82	128	-0.19	0.85
Number of physio visits 5-6	-0.85	-5.05 to 3.34	128	-0.4	0.69
Number of physio visits 7-8	-0.35	-4.54 to 8.84	128	-0.16	0.86
Number of physio visits 9-10. Reference group	0		.	.	.
Six-week follow-up	0.66	-1.40 to 2.73	113	0.63	0.52
12-week follow-up. Reference group	0				
Age	-0.00	-0.13 to 0.12	128	-0.1	0.91
Gender female	1.99	-0.71 to 4.69	128	1.44	0.15
Gender male. Reference group	0		.	.	.
Unemployed	-0.19	-4.98 to 4.59	128	-0.08	0.93
Employed. Reference group	0		.	.	.
Education: university/post grad.	3.20	-0.06 to 6.48	128	1.92	0.06
Education: Diploma	1.72	-1.33 to 4.78	128	1.1	0.27
Education: Completed school. Reference group	0		.	.	.
Smoker: no	-0.03	-2.77 to 2.71	128	-0.02	0.98
Smoker: yes. Reference group	0		.	.	.
BMI	0.03	-0.21 to 0.28	128	0.28	0.78
Pain Duration	-0.06	-0.23 to 0.10	128	-0.74	0.45
Treatment PW x Neuropathic Pain phenotype	1.81	-4.16 to 7.78	128	0.59	0.55
Treatment PW x Nociceptive Pain phenotype. Reference group	0		.	.	.
Treatment P x Neuropathic Pain phenotype	-1.41	-7.70 to 4.86	128	-0.44	0.65
Treatment P x Nociceptive Pain phenotype. Reference group	0		.	.	.

Effect	Estimates	Confidence intervals	df	t	p value
Treatment W x Neuropathic Pain phenotype. Reference group	0		.	.	.
Treatment W x Nociceptive Pain phenotype. Reference group	0		.	.	.
Treatment PW x Six-week follow-up.	0.10	-2.55 to 2.75	113	0.07	0.94
Treatment PW x 12-week follow-up. Reference group	0				
Treatment P x Six-week follow-up.	0.26	-2.43 to 2.96	113	0.19	0.84
Treatment P x 12-week follow-up. Reference group	0				
Treatment W x Six-week follow-up. Reference group	0				
Treatment W x 12-weeks. Reference group	0				
Neuropathic pain phenotype x Six-week follow-up.	-0.30	-2.51 to 1.89	113	-0.27	0.78
Neuropathic pain phenotype x 12-week follow-up.. Reference group	0				
Nociceptive pain phenotype x Six-week follow-up. Reference group	0				
Nociceptive pain phenotype x 12-week follow-up. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

Figure 16 demonstrates the changes in PCS score within treatment groups between baseline and follow-up at six-weeks and 12-weeks. The linear mixed model regression analyses showed there was no statistically significant effect of time (six-weeks and 12-weeks follow-up), treatment group allocation, or interaction between treatment group allocation and time.

Figure 16: Comparison of PCS score within each treatment group over time.



P, usual care treatment; **W,** pedometer-based walking intervention;

PW, usual care treatment and pedometer-based walking intervention;

Week 0, baseline; **Week 6**, six-week follow-up; **Week 12**, 12-week follow-up

4.6 Additional measures

Expectation of pain intensity following treatment at 12-week follow-up and walking data are presented below.

Participant expectation for pain intensity

Data was not normally distributed therefore median scores were described. There was no missing data in this section. The statement was anchored with 0= no pain and 100= worst imaginable. Baseline scores were 10/100 (Table 37).

Spearman's rank correlation was used to explore the relationship between expected pain intensity at baseline and change in pain intensity between baseline and 12-week follow-up in the entire cohort. There was a strong correlation with expected pain intensity and actual change in pain intensity at 12-week follow-up (p=0.01). This suggests that those who expected lower pain intensity had lower pain intensity at the 12-week follow-up, compared to those who expected less improvement who had low improvement. The strength to this relationship was moderate (r=0.27).

Walking data

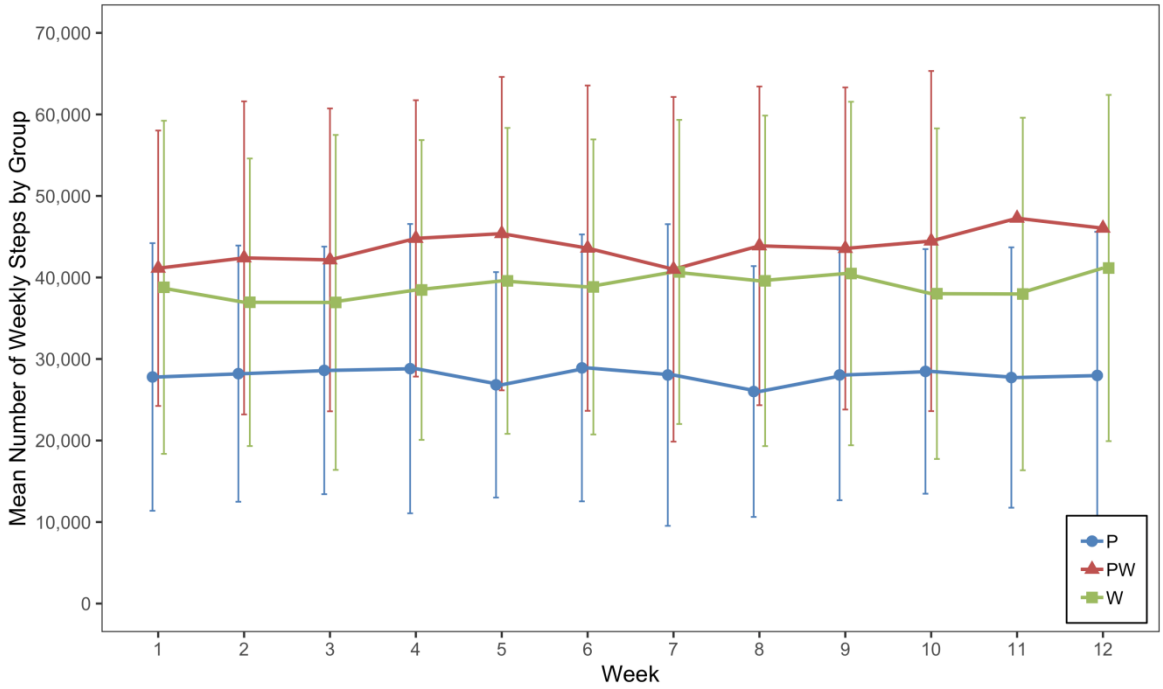
Daily steps walked and distance covered was recorded across all three treatment groups, using a pedometer. Data was recorded in the provided participant study diary. In addition, participants in the W and PW treatment groups recorded number of steps taken, distance covered, and time taken to perform the walking intervention.

There was missing walking data for one participant for weeks 1-6; (diaries not received). In this case no baseline observation carried forward (BOCF) substitution was possible. Walking data were presented as mean weekly steps for all treatment groups over the 12-week intervention. Steps referred to in the data were separated into walking intervention steps and mean daily steps. Week 0 = baseline and week 1 = data at the end of the first week. Since walking as a physical activity could be measured by steps and minutes spent doing the walking intervention, the W and PW treatment groups walking intervention data were presented together to eliminate any differences in pacing. The effect of treatment group, week in the study, and pain phenotype on total number of weekly steps taken was analysed using a linear mixed model.

Total weekly steps

The number of weekly steps by treatment group is illustrated in Figure 17.

Figure 17: Mean number of weekly steps at the end of each week, by treatment group



Data presented as mean (Error bars denote SD).

PW: Usual care and walking intervention

W: Walking intervention only

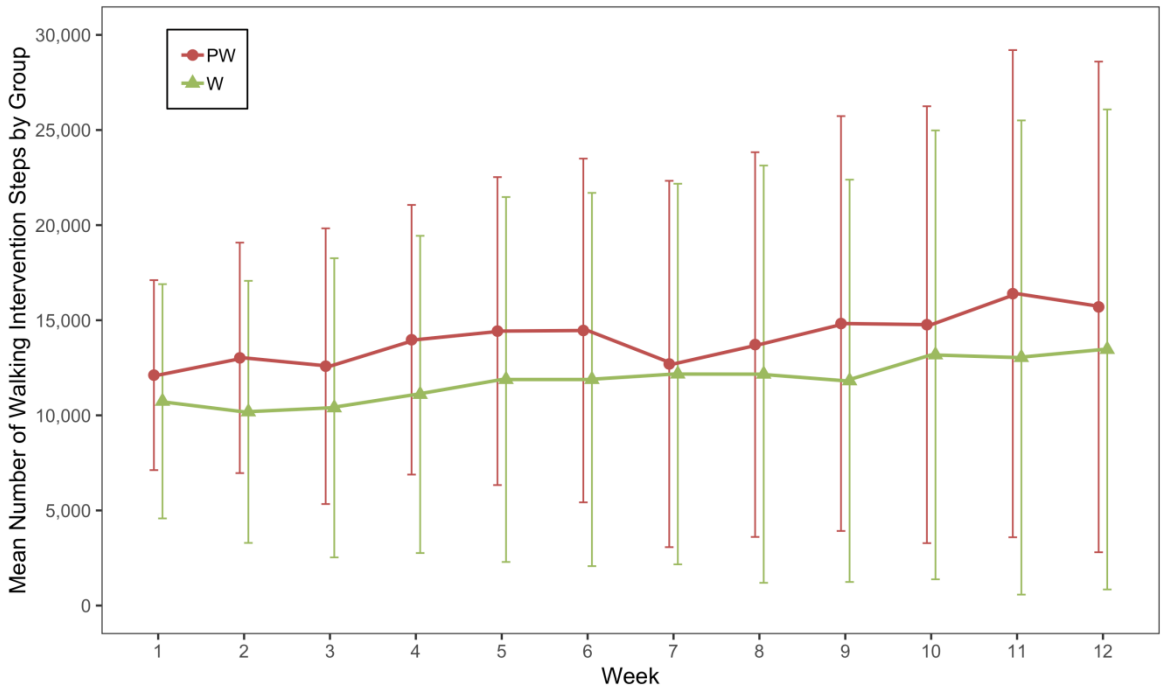
P: Usual care only

Both W and PW treatment groups received the same, paced, and progressive walking intervention. The total weekly steps were greater in these two treatment groups versus the P group, who self-paced. The P group achieved fewer steps than the W and PW groups throughout the 12-weeks.

Total weekly steps: Walking intervention

The walking intervention was designed to increase time spent walking by 10% per week. These data represent walking intervention steps separate from total weekly steps. The mean number of walking intervention steps per week by treatment group is illustrated in Figure 18. The trend shows a complementary pattern to data in total weekly steps.

Figure 18: Mean number of steps per week in usual care and walking intervention treatment group and the walking intervention treatment group only.



Data presented as mean (Error bars denote the SD).

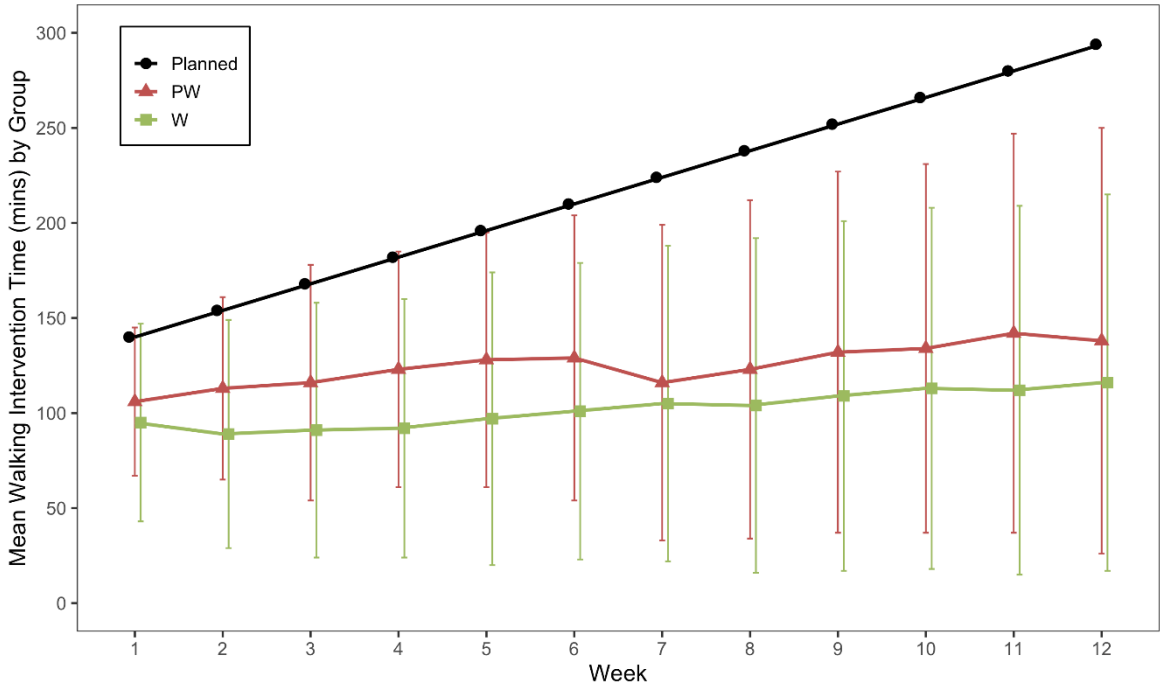
PW: Usual care and walking intervention

W: Walking intervention only

Total weekly time spent doing the walking intervention

The mean walking intervention time in minutes, for each treatment group with a walking intervention is illustrated below (Figure 19). Both intervention groups using walking as exercise executed fewer minutes walking than planned in the treatment.

Figure 19: Mean number of minutes walked, by group allocation, per week.



Data presented as mean (Error bars denote the SD).

PW: Usual care and walking intervention

W: Walking intervention only

Comparison of walking data between treatment groups

Total weekly steps by treatment group were recorded for 12 weeks. To interpret average steps per day, mean weekly steps were divided by seven (Table 50).

Table 50: Summary table demonstrating total number of weekly steps taken by treatment group.

TREATMENT GROUP	Week	Number of observations	Mean weekly steps	Std Dev	Minimum	Maximum	Mean weekly steps divided by 7 days	Average steps per day
P	1	46	27 796	16 413	2 149	78 253	3970	3993
	2	46	28 203	15 715	580	81 177	4029	
	3	46	28 601	15 185	1 787	77 741	4085	
	4	46	28 816	17 746	1 887	102 486	4116	
	5	46	26 833	13 827	2 556	56 933	3833	
	6	46	28 908	16 372	3 896	84 594	4129	
	7	46	28 035	18 503	3 696	81 338	4005	
	8	46	26 010	15 383	1 866	73 639	3715	
	9	46	28 039	15 368	2 149	71 589	4005	
	10	46	28 476	15 004	1 195	72 669	4068	
	11	46	27 726	15 970	1 934	69 889	3960	
	12	46	27 982	17 619	2 149	69 703	3997	
PW	1	48	41 132	16 892	7 280	82 708	5876	6258
	2	48	42 401	19 204	5 061	96 506	6057	
	3	48	42 159	18 575	7 280	84 485	6022	
	4	48	44 792	16 951	7 280	94 485	6398	
	5	48	45 380	19 223	7 280	94 300	6482	
	6	48	43 594	19 955	7 280	88 443	6227	
	7	49	41 005	21 140	0	105 678	5857	
	8	49	43 879	19 549	7 280	81 870	6268	
	9	49	43 556	19 757	7 280	79 567	6555	
	10	49	44 467	20 862	7 280	86 659	6352	
	11	49	47 267	23 756	7 280	98 228	6752	
	12	49	46 043	25 388	2 933	111 672	6577	
W	1	52	38 796	20 431	1 459	112 921	5542	5565
	2	52	36 957	17 637	1 459	79 136	5279	
	3	52	36 939	20 546	1 459	104 706	5277	
	4	52	38 467	18 388	1 459	74 821	5495	
	5	52	39 582	18 762	1 459	83 168	5654	
	6	52	38 839	18 095	1 459	78 358	5548	
	7	52	40 679	18 656	1 459	83 542	5811	
	8	52	39 586	20 273	1 459	88 074	5655	
	9	52	40 483	21 069	1 459	83 150	5783	
	10	52	38 011	20 276	1 459	89 611	5430	
	11	52	37 972	21 622	1 459	90 465	5424	
	12	52	41 157	21 236	1 459	94 997	5879	

A linear mixed model was generated to determine the effect of treatment group allocation, pain phenotype and time on total number of weekly steps (Table 51).

Table 51: Linear mixed model 9 - fixed effects output exploring contributions of treatment group allocation, number of weeks in the study (1-12), and pain phenotype on total weekly steps.

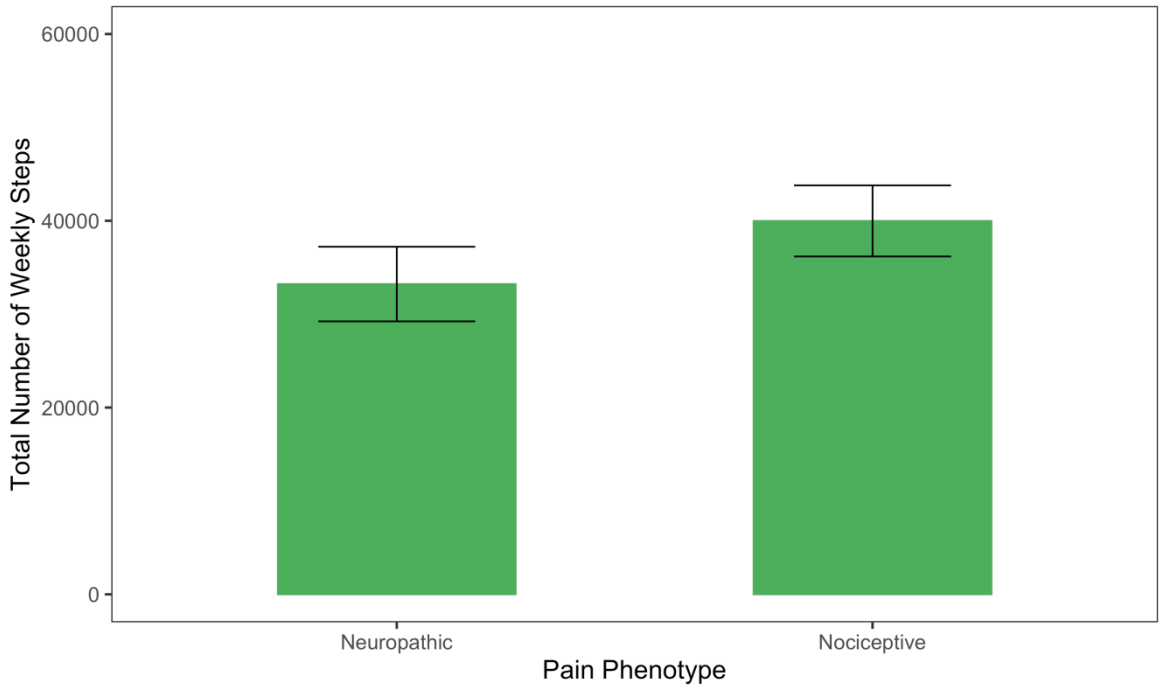
Effect	Estimate	Confidence intervals	df	t	p value
Intercept	46048	39570.16 to 52525.84	189	13.93	<.01
Treatment PW	2644.17	-6892.41 to 12180.75	176	0.54	0.58
Treatment P	-12831	-22480.5 to -3181.51	176	-2.61	0.01**
Treatment W. Reference group	0				
Week 1	-3652.52	-7393.04 to 88.00	1027	-1.91	0.06
Week 2	-5475.48	-9216 to -1734.96	1028	-2.87	0.01**
Week 3	-4935.47	-8675.89 to -1195.05	1028	-2.59	0.01**
Week 4	-3758.13	-7499.67 to -16.588	1030	-1.97	0.05*
Week 5	-2701.51	-6442.64 to 1039.62	1034	-1.42	0.15
Week 6	-3131.84	-6870.21 to 606.52	1044	-1.64	0.10
Week 7	-1662.87	-5398.69 to 2072.94	1070	-0.87	0.38
Week 8	-2607.75	-6328.26 to 1112.76	1135	-1.37	0.16
Week 9	-1352.93	-5031.69 to 2325.83	1288	-0.72	0.47
Week 10	-4065.27	-7604.76 to -525.78	1529	-2.25	0.02*
Week 11	-2751.71	-5870.15 to 366.72	1245	-1.73	0.08
Week 12. Reference week	0		.	.	
Neuropathic pain phenotype	-11858	-21446 to -2270.01	163	-2.42	0.02*
Nociceptive pain phenotype. Reference group	0				

p-value < 0.05: *; p-value < 0.01: **; p-value < 0.001: ***.

The total number of weekly steps for the P group were significantly lower than that for the PW treatment group (p=0.01) (Table 51). Weeks two, three, four and ten in the study had a statistically significant effect on total weekly steps taken compared to the reference group, week 12 (p=0.01, p=0.01, p=0.05 and p=0.02 respectively). Given the wide confidence intervals, at every time point it is unclear whether or not the significant findings are spurious. The trend for walking to increase over the 12-week intervention period is demonstrated in figure 17.

There was a statistically significant effect of pain phenotype on total weekly steps (p=0.02), such that participants with a nociceptive pain phenotype, accumulated a greater total number of weekly steps than did participants with a neuropathic pain phenotype (Table 51, Figure 20). No other variables in the model predicted total weekly steps walked.

Figure 20: Weekly total step number of nociceptive and neuropathic pain phenotypes.



Chapter 5: Discussion

5.1 Introduction

At the time of writing, this was the first RCT where the aim was to compare the effect of a pedometer-based walking intervention; usual care physiotherapy and a combination of both treatment groups on pain intensity and associated biopsychosocial outcome measures in participants with nociceptive and neuropathic chronic lower back pain (CLBP).

The three treatment groups used in this RCT are independently recognized in previous studies included in reviews to improve mean pain and disability outcome scores using walking and physiotherapy treatments (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

The primary outcome was change in global pain intensity from baseline to 12-week follow-up as measured by participants on a NRS. Secondary outcomes were changes in disability, kinesiophobia and catastrophic thinking over the same time period measured using the ODI, TSK and PCS.

The main finding of this RCT concluded that there were no statistically significant differences in pain intensity, disability, kinesiophobia and pain catastrophizing between baseline and 12-week follow-up between the three treatment groups. The findings in the present RCT concurred with those in previous trials treating CLBP where no statistically significant difference in pain intensity between groups was observed when comparing walking to exercise groups or walking to usual care (Eadie et al., 2013; Hurley et al., 2015). The research question therefore could not be answered with no statistically significant differences between treatment groups. However, a minimum clinical important difference in reduced pain intensity is observed in the PW treatment group at 12-week follow up.

The findings in the current RCT showed significant improvements in pain intensity, disability, kinesiophobia and pain catastrophizing over time in all three treatment groups. It has been said that participation in a trial with intensive assessment and monitoring received from researchers can improve the course of symptoms (Artus et al., 2010). This could be related to placebo or regression to the mean. With multiple levels of influence, general improvement in CLBP may be related to seeking and receiving care regardless of treatment.

Attending more than two physiotherapy visits across groups over the intervention period, demonstrated significantly improved pain, kinesiophobia and pain catastrophizing outcome measure scores between baseline and 12-week follow-up, regardless of group allocation. This dosage of treatment affecting outcomes has yet to be explored in the literature exploring walking and physiotherapy treatments for CLBP thus far.

The three treatment groups used in this RCT have to date not yet been compared by recording PA by measuring weekly steps in every treatment group over the intervention period. Participants randomised to walking interventions (W and PW) increased their weekly steps from baseline when compared to the usual care physiotherapy treatment group whose weekly steps remained consistent. Regardless of PA changes due to treatment group allocation, treatment group allocation did not predict changes in outcomes. According to the advised walking used in the current trial, additional walking exercise when combined with usual care physiotherapy did demonstrate the greatest improvement in mean pain intensity score even though there was no statistically significant difference in pain intensity. This potentially indicates minimal clinical change in pain intensity when walking is combined with physiotherapy.

There is a growing body of evidence highlighting pain heterogeneity (Chetty et al., 2012; Baron et al., 2016). The current trial did not examine pain phenotype between treatments however due to the focus being on the primary outcome of pain intensity, it was deemed salient that the modelling process involved phenotyping pain in the CLBP cohort. To date, when studying pain intensity, pain phenotypes of CLBP patients in physiotherapy cohorts have not been widely explored. In this RCT, each patient's predominant phenotype was identified using the painDETECT questionnaire, their symptom burden determined and the association between pain phenotype and the outcome measures were studied by including pain phenotype in the modelling process. Due to the confounding factors of pain intensity with pain phenotype, and disability with pain phenotype, it is unclear whether pain intensity or pain phenotype, and disability or pain phenotype were predictive of pain intensity and disability at 12-week follow-up. This may explain limited attempts to study outcomes associated to pain phenotypes when examining CLBP treatments. In the entire cohort, participants with neuropathic pain phenotype took fewer steps over the course of the RCT compared to those with nociceptive pain phenotype. The current trial in South Africa concurred with international studies looking at pain phenotype demonstrating that the clinical burden (increased pain and disability), was greater in patients with a neuropathic phenotype compared to those experiencing nociceptive pain (Smart et al., 2012b; Baron et al., 2106; Spahr et al., 2017).

Seen in reviews on walking and physiotherapy as a CLBP treatment, the pain findings of this RCT are comparable to studies where walking as an exercise was shown not to be statistically significantly

different to treatment using physiotherapy, pain education, or exercise groups in previous reviews (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). However, a clinical significance was observed in reduced pain intensity scores when combining walking and physiotherapy. A salient finding demonstrates that the number of physiotherapy visits is an important clinical variable to consider in CLBP outcome measurement change.

5.2 Assessment of trial strengths and limitations

Strengths

The interventions have each individually been compared to other interventions, but never to each other (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). The literature up until now demonstrates each individual treatment groups application. The current RCT demonstrates the largest mean score changes in all outcome measures in the PW treatment group although no statistically significant differences between groups in changes in outcomes were observed. Due to the usual care physiotherapy component typically used, it is highly relevant in physiotherapy in South Africa and the UK (Naidoo et al., 2012; National Institute for Health and Care Excellence, 2016). The strength of this RCT is within the context of physiotherapy treatment decision in a specific organizational infrastructure. Depending on the patient's needs, and what the physiotherapist can deliver, these three treatments demonstrate small improvements in all outcomes although physiotherapy combined with walking exercise shows the largest mean score improvements in all outcome measures. Patients and clinicians have an informed choice as to which treatment to offer due to the results from this RCT.

This RCT demonstrated that the number of visits to the physiotherapist requires consideration to achieve significant change when treating CLBP outcome measures used in this RCT. The results indicate that three or more (up to ten) physiotherapy visits will change these outcomes when using either of these treatment groups. Physiotherapists and patients may now be aware that a minimum exposure may be necessary to achieve significant changes when treating CLBP over 12-weeks.

There is a paucity of evidence examining pain catastrophizing when comparing walking to physiotherapy treatments. The current RCT demonstrates that reductions in pain catastrophizing were observed when using the three treatments in this RCT. Placebo or regression to the mean however can also explain the improved PCS score.

Additionally, it is the first time that each intervention used in this RCT measured step counts in the same trial. The results show that usual care physiotherapy did not increase step count PA over twelve weeks, however the two other walking intervention treatment groups did. This objective comparison

together with the results showing no significant difference between treatment groups outcomes may influence physiotherapist decisions not to increase patient step counts per se, but to concentrate on education of CLBP and patient interaction. Notwithstanding, a clinically meaningful reduction in pain intensity was seen in the PW treatment group at the 12-week follow-up. The limited increase shown in PA in the PW treatment group has merit for exploring combining a partly supervised pedometer-based walking program to usual care physiotherapy when treating pain intensity.

Results demonstrated that delayed progress is to be expected with patients with a neuropathic pain phenotype in all outcomes used. There is a lack of certainty as to which treatments to offer patients with CLBP having a neuropathic pain phenotype (Chetty et al., 2012; Naidoo et al., 2012; Clenzos, Naidoo and Parker, 2013; Baron et al., 2016). Because this trial was not powered for pain phenotypes, uncertainty remains for specific physiotherapy treatments benefitting pain phenotype.

There is a paucity of studies of CLBP treatment executed in Africa (Louw, Morris and Grimmer-Somers, 2007; Morris et al., 2018). Conducting the study in Africa demonstrates improved knowledge related to CLBP in an African context.

The intervention period of 12-weeks in this RCT is a strength, since trials should be ideally 12-weeks long (Moore and Wiffen, 2013). This length was observed in one RCT comparing three treatment groups; walking as self-exercise, an exercise group using strength and flexibility, and a physiotherapy group (Torstensen et al., 1998). RCTs using walking to treat CLBP had different lengths of interventions including eight weeks (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015), six weeks (Shnayderman and Katz-Leurer, 2013) and four weeks (Karadeniz et al., 2014). Duration bias possibly overestimating the effectiveness of the intervention, may be seen in the trials with these interventions shorter than 12-weeks (Moore and Wiffen, 2013). Interventions showing improved outcomes all were relatively short (Eadie et al., 2013; McDonough et al., 2013; Shnayderman and Katz-Leurer, 2013; Karadeniz et al., 2014; Hurley et al., 2015). Results in two similar RCTs concurred with that of the current RCT (Eadie et al., 2013; McDonough et al., 2013). Showing no significant difference between treatment groups in the current RCT may suggest shortening interventions from 12 weeks to eight weeks.

Furthermore, none of these mentioned trials demonstrated a MCID of >2/10 for improvements in pain intensity (Eadie et al., 2013; McDonough et al., 2013; Shnayderman and Katz-Leurer, 2013; Karadeniz et al., 2014; Hurley et al., 2015). There was a mean difference of -26.9 in a 0-100 NRS in the current trial PW treatment group. The confidence intervals of -35.9 to -17.9 demonstrate this was a plausible minimal clinical change. Three other previous trials showed improvements in mean pain intensity score >2/10. Interventions of lumbar traction and walking (Mirovsky et al., 2006), home

exercise combined with walking, compared to both physiotherapy, and home exercise (Koldaş Doğan et al 2008), walking exercise, education on CLBP, and strength exercise compared to motion control exercise combined with strength and stretching exercises (Magalhães et al., 2015) all showed improved mean pain intensity MCIDs >2/10 at follow-up. Follow-ups however were all less than six weeks. The MCID shown in the PW group in the current trial according to Moore and Wiffen (2013), did not include the duration bias. If the PW treatment group showed statistical and clinically significant differences, the intervention could wholly be considered effective.

Limitations

The efficacy of the treatment groups may only be truly explored if a true control (no intervention) was added to this RCT. No treatment group was used with absent treatment.

Unlike other studies using walking and physiotherapy interventions, which examined the longer-term effects on pain outcomes and the sustainability of the effects, only a 12-week follow-up was completed in the current RCT. In RCTs using walking to treat CLBP, a 6-month follow-up point was used (McDonough et al., 2013; Eadie et al., 2013; Krein et al., 2013; Hurley et al., 2015). Furthermore, three RCTs used year follow-up (Torstensen et al., 1998; Krein et al., 2013; Hurley et al., 2015). A follow-up of the participants may also have allowed for observation of long-term effects on outcomes and any behaviour change regarding weekly step counts. With pain intensity as the primary outcome, the outcomes disability and fear avoidance behaviour followed-up at one year is beyond the scope of this discussion. At six months, two feasibility studies measured average pain on an NRS in all treatment groups (McDonough et al., 2013; Eadie et al., 2013). Average pain improved in all other interventions but did not reduce in only the walking intervention in Eadie et al., (2013). At six months two RCTS demonstrated NRS average pain scores had improved significantly, with no difference between pain scores between treatment groups in each RCT (Krein et al., 2013; Hurley et al., 2015). At one year there were significant improvements in pain scores with no differences between groups (Hurley et al., 2015). At one year, although both groups pain intensity continued to be lower than baseline, these differences were no longer significant (Krein et al., 2013). The Norwegian study at one year indicates that although pain in the lower back was not significantly different between groups, pain in physiotherapy and medical exercise was significantly lower than in the self-exercise walking group (Torstensen et al., 1998). Although not expressing pain phenotypes in this RCT, behaviour of referred pain typical of neuropathy appears to present differently to localized pain after one year (Torstensen et al., 1998). There appears to be a time effect on the interventions beyond 12-weeks, although the sustainability of the intervention effects on pain intensity in the current RCT without a longer-term follow-up cannot be commented on.

The American study which compared pedometer readings of two groups, one with an internet supported walking program with goals and the other without a walking program showed significant differences between groups at six months, but not so at one year follow-up (Krein et al., 2013). The group randomised to the walking program showed statistically significant more average daily steps between baseline and six months only (Krein et al., 2013). Furthermore, at six months pedometer data substantiated increased PA in the walking and education group versus the education alone group in McDonough et al., (2013). Behaviour in increased walking appears to affect these CLBP treatment groups up to six months (Krein et al., 2013; McDonough et al., 2013). The behaviour in increased walking in walking interventions used in the current trial cannot be commented on at six months.

There is a recent move internationally to offering Pain Neuroscience Education (PNE) to patients with CLBP (Puentedura and Flynn, 2016; Hush et al., 2018). The reviews on using walking to treat CLBP involve education on CLBP in four RCTs (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015; Magalhães et al., 2015). Although education and support were provided in the current RCT, it was not standardized as is documented in "the Back Book" (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015; Magalhães et al., 2015). It was also standardized in the 5A's methodology (McDonough et al., 2013). This RCT would have been more repeatable if a standardized education and support protocol were used when education on CLBP was provided to all participants.

Unlike the current RCT, self-efficacy was measured in other walking interventions (Krein et al., 2013; McDonough et al., 2013, and Hurley et al., 2015). To predict PA and coping strategies, self-efficacy can be measured with several scales and is documented in a meta-analysis and review (Brady, 2011; Jackson et al., 2014). It would be of use to include self-efficacy or the Patterns of Activity Measure-Pain (Huijnen et al., 2011). The use of these measures may have explained pacing differences in the W and PW groups as the weekly step count in the current RCT did not increase weekly as may have been anticipated by the methodology set out for the walking intervention.

A limitation in the recording of data using step counts is noted. Recording step counts prior to baseline, using a minimum of three days per week rather than one day per week of step counts to calculate weekly averages, and presenting data as steps per day rather than weekly were limitations in the current trial. Recording weekly step counts in all three groups prior to receiving any interventions in the current RCT would have been preferable in order to objectively include those insufficiently active. On the assumption that all three treatment groups began with a similar weekly step count due to the subjective inclusion criteria of being insufficiently active, after one week of advised pedometer walking, the W and PW group exceeded the step count of the P group. A solution to this would be to recruit and complete baseline measures, and only introduce a walking intervention

a week later. For the week prior to introducing the walking interventions, the weekly step count could have been recorded. One RCT was more specific and successful in recording activity and step data (McDonough et al., 2013). The same RCT was more specific in calculating pedometer data and change in steps per day from baseline as well as adherence to the walking program. The researchers cross checked their step count data to exclude long periods of time where steps were not recorded (McDonough et al., 2013). Furthermore, the RCT recorded baseline step data prior to beginning the walking program objectively to ensure insufficiently active behaviour of included participants (McDonough et al., 2013). Concurring with the Irish study, further thought needs to be given in measuring PA. In the current RCT, daily steps which were not entered were removed from the weekly calculation. For example, if seven days steps were completed the weekly amount would be divided by seven. If only two days steps were recorded, the amount would be divided by two. The current RCT design did not account for steps taken on days which were not recorded in participant diaries and therefore daily step variability may require further exploration. Accordingly, minimum valid step recorded should be three days per week (Tudor-Locke et al., 2011). The current trial minimum was one day per week. The aim of this trial however was not an association of outcomes to steps per week or day, but to intervention treatment groups. At the time of design, the current RCT priority was to compare objective steps per week and not use steps per day for analysis as the comparison between treatment groups in analysis was through weekly steps and not daily steps.

This RCT did not record all adverse events related or unrelated to the walking program as was done in other RCTs (McDonough et al., 2013; Hurley et al., 2015; Krein et al., 2013). Only some participants who discontinued interventions reasons were recorded at baseline (P n=6, PW n=3, W n=13). Those lost to follow-up did not provide reasons (P n=7, PW n=4, W n=7). In total, 27% of participants had dropped out by 12-week follow-up and appears to be in the range of related trials using walking and physiotherapy to treat CLBP patients. Adverse effects were seen in 35% of participants in McDonough et al., (2013), 27% in Krein et al., (2013) and 19% in Hurley et al., (2015). Adverse events in these trials included colds and flu, road traffic collisions, ankle sprains, increased LBP, allergic reaction to the metal pedometer, callouses and chest pain. Despite 35% of participants having adverse events in one RCT, only one related adverse event led to the patient stopping the walking program (McDonough et al., 2013). Additionally, in the current RCT, participants were not encouraged to record as to why they were not able to maintain incremental time to walk in the W and PW groups. If this data had been captured and analysed in the current RCT, potential obstacles to using the interventions, improved goal setting and improved pacing for a walking intervention may have been demonstrated.

Different content, context and organizational infrastructure of health care systems is salient when comparing implementation of treatments. This study was done in private practice in South Africa

using a UK NHS NICE model for treatment. However, a study not using walking as a treatment, comparing LBP physiotherapy treatment in public and private practice physiotherapy showed similar outcomes at follow-up (Casserley-Feeney et al., 2012). Furthermore, comparing data from different countries can be problematic with different social structures. Factors including country, and private versus public organisational infrastructure vary in the studies used in comparison in this RCT. This does not reflect the heterogeneity of populations suffering with CLBP within different countries and health systems.

No full pilot study was conducted. The feasibility study in the current RCT lacked a template for data entry, therefore no analysis could be done at early stages. The necessary components for data entry and analysis were only executed with the main RCT. This led to a lengthy period of data collection and analysis.

This was the first trial using walking and physiotherapy to treat CLBP using pain phenotyping in the modelling process. The cut-off points in the painDETECT for neuropathic pain used previously, have been scores of 19-38 (Spahr et al., 2017). In the current RCT scores 13-38 were used as cut-off points for neuropathic pain (values 13-17 were defined in the tool as ambiguous, "likely having a neuropathic component"). The current RCT therefore included the ambiguous range as part of the neuropathic pain phenotype. This would mean that some participants were included in the neuropathic pain phenotype that may have had a greater proportion of a nociceptive pain phenotype. As agreed in a review on neuropathic LBP in clinical practice, a more rigorous approach would be to combine the screening tool with a focused clinical examination (Baron et al., 2016). One could include scores 19-38 on painDETECT to only categorize those likely having a neuropathic component. The trial was also not powered for differences between pain phenotypes within treatment groups. Clinical use could be made if analysis could indicate differences between treatment groups applied to specific pain phenotypes.

5.3 Comparison of the findings to other studies

Baseline findings described similarities and differences in this cohort to existing CLBP literature. This cohort presented with higher levels of education and greater employment levels, unlike previous epidemiological studies on CLBP (Rubin et al., 2007; Schwellnus et al., 2011). Most of the cohort were high school educated, with 31% having a diploma and 29% a university degree; and 92% were employed. This difference in demographics might have influenced participants in this study to choose private physiotherapy practices, which attracts patients with more disposable income and higher levels of education, as evidenced in an African study comparing private and public medical practice patient demographics (Matchaya and Muula, 2009). Little is known about these differences in South

African physiotherapy practices, although the South African Society of Physiotherapy is underway in developing data strategies to improve comprehensive service for all communities (Fourie, 2019). Observed in this RCT, more women experience CLBP, and participants have increased BMIs. This too was observed in epidemiological studies of CLBP (Rubin et al., 2007; Schwellnus et al., 2011). It was possible to distinguish pain phenotypes at baseline in the cohort.

Primary outcome measure: pain intensity

Unlike the current RCT, patients' pain intensity had not been previously phenotyped in other trials used in reviews comparing walking to other physiotherapy interventions (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). Before exploring the effects of independent variables included in the RCT, pain intensity at baseline was found to be a confounding variable with pain phenotype. In order to examine the contributing effect of both of these variables on pain intensity at 12-week follow-up, models were constructed either controlling for pain intensity at baseline and excluding pain phenotype or controlling for pain phenotype and excluding pain intensity at baseline. In addition to this association at baseline, the models found neuropathic pain phenotype to be a predictor of greater pain intensity at 12-week follow-up too. Unlike patient assessments observed in the physiotherapy and walking literature over a decade ago, this more specific evaluation of the patient concurs with the move in 2016 NHS NICE guidelines to further stratify patients with CLBP and underlying neurophysiological phenotypes. Recognizing these phenotypes may elucidate neurophysiological differences within cohorts.

The fundamental finding in the current RCT was that the NRS for pain intensity was not predicted due to treatment group, with no statistically significant between group differences. The findings in the present RCT concurred with those in previous trials treating CLBP where no statistically significant difference in pain intensity between groups was observed when comparing walking to exercise groups or walking to usual care (Eadie et al., 2013; Hurley et al., 2015). In this RCT, pain intensity improved significantly from baseline to 12-week follow-up, within all three treatment groups. Thus, pain intensity improved regardless of treatment group. The research question could not be answered with no statistically significant differences between treatment groups. A meta-analysis of 70 RCTs (included 57% CLBP participants) and 19 cohort studies (included 11% CLBP participants), found that the clinical course of LBP symptoms including pain intensity improved regardless of the treatment (Artus et al., 2010). Similarly, the result of no statistically significant between group differences was seen in two RCTs comparing walking interventions to usual care physiotherapy and supervised exercise classes in CLBP patients, where all three treatment groups showed improved pain intensity scores (Eadie et al., 2013; Hurley et al., 2015).

However, when treating patients with pain intensity as an outcome a minimally clinically important difference is considered salient in judging the clinical application of a therapeutic regimen (Page 2014). Concurring views show that a 2.0 reduction on 0-10 NRS is recognized as a minimal clinically important difference (MCID) on the NRS (Farrar et al., 2000; Farrar et al., 2001; Haefeli and Elfering, 2006; Suzuki et al., 2020). In the current RCT when examining mean change from baseline to follow-up points in pain intensity scores, the greatest mean score change between three treatment groups is in the partly supervised pedometer walking program combined with usual care physiotherapy (-26.9/100) despite no statistical significance between groups. The confidence intervals of -35.9 to -17.9 indicate that there is a plausible minimal clinical change. When observing the mean change from baseline scores for pain intensity and between-group effect sizes, the effect size was moderate. The only other trial using the combination of usual care physiotherapy and walking exercise, comparing usual care physiotherapy and an over ground walking intervention, with physiotherapy and a treadmill walking intervention, demonstrated statistically significant improvements in pain intensity scores in both treatment groups (Karadeniz et al., 2014). Although these treatments were similar to the PW treatment group in the current RCT, no mean change in pain intensity scores were provided so understanding if a MCID was achieved is unknown.

Three trials in Table 19 comparing walking to physiotherapy modalities used on patients with CLBP demonstrate a potential MCID in pain intensity (Mirovsky et al., 2006; Koldaş Doğan et al 2008; Magalhães et al., 2015). Follow-ups in these three studies were at six weeks or less. The remaining trials in Table 19 with follow-ups eight weeks or longer reported smaller improvements in pain intensity. The results in the current RCT and a follow-up at 12-weeks support further investigation into the clinical application of a partly supervised pedometer-based walking program added to usual care physiotherapy.

Number of physiotherapy visits

The number of physiotherapy visits predicted reduced pain intensity at 12-week follow-up in all treatment groups. There is limited evidence around the number of physiotherapy visits affecting outcomes. There is a wide range of number and types of physiotherapy interactions in other studies. In the reviews using walking to treat CLBP, the heterogeneity in number of visits or supervision was not measured as an independent variable (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). The current RCT demonstrated that three or more treatments show significant reductions in pain. Differences in the way physiotherapy visits or interactions are provided in previous studies have been observed. Physiotherapy visits include telephone, internet, and face to face consultation. The common factors which appear in visits are the provision through these visits of support for participants and/ or education on how to manage their pain.

In this RCT the number of physiotherapy visits within each of the three treatment groups varied. This was due to combined patient and physiotherapist choice and was similar to that found in other studies (Eadie et al., 2013; Hurley et al., 2015). The ideal number of physiotherapy visits for CLBP has not been extensively studied and visit numbers vary considerably in the literature. The current RCT is the first which included the number of physiotherapy visits as an independent variable in the models used to determine pain intensity, when compared to the studies in systematic reviews on walking interventions for CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

There is however an array of information on the variety of numbers of physiotherapy visits for LBP studies which includes both acute and CLBP. Physiotherapy visit data is scarce and due to differences in parametric and non-parametric data, findings up to now are expressed with both mean and median values. A retrospective study of 64 CLBP patients with symptomatic grade I spondylolisthesis evaluated the optimal number of physiotherapy visits needed to improve pain outcomes (Ferrari et al., 2018). Patients were divided into two groups; those who had a mean number of five - eight physiotherapy visits, and those who had nine - 12 visits. Both groups received similar stabilising exercises, massage, and pain education in a six - 16-week intervention. Both groups had similar NRS outcomes, indicating superior efficiency when using five – eight physiotherapy visits when treating spondylolisthesis CLBP pain intensity. In a nationwide survey of LBP treatments conducted in outpatient practices in the USA, a mean number of 11 physiotherapy visits over five weeks was observed (Jette et al., 1994). A median number of five visits (range 0-20 visits) over a median number of four weeks (range 1-19 weeks) were observed in a Northern Ireland CLBP study (Gracey et al., 2002). Two studies in the Netherlands showed varied mean number of treatments, with van Baar et al., (1998) demonstrating seven treatments (range 6-15 weeks), compared with mean (S.D) 9.9 (6.6) treatments in 1733 NSLBP patients (Swinkels et al., 2005). The treatment visits ranges varied extensively (minimum 1; maximum 67) (Swinkels et al., 2005). In a survey of 846 Canadian physiotherapists, the majority of patients with LBP, mostly in private practice, would visit the physiotherapist between two to three times per week for one to three months (Orozco et al., 2017). Despite use of mean and median data, the range of physiotherapy visits used clinically is large. Despite the large range, the current RCT suggests a minimum number of visits necessary though for a statistically significant change in pain intensity.

Varied lengths of treatment in a chronic condition like CLBP may be the reason for the paucity of evidence on an optimal number of physiotherapy visits for CLBP. In studies where walking has been used as an intervention for CLBP, a variety of number of physiotherapy visits are demonstrated between studies with almost all interventions showing reductions in pain intensity. For example, comparing supervised exercise, usual care physiotherapy and a self-paced walking intervention, all

participants barring those in the walking intervention (who received phone calls every second week), received 36 face to face physiotherapy treatments (three per week for one hour over 12 weeks) (Torstensen et al., 1998). Traction with a walking program was compared to traction alone, where each patient received 20 face to face physiotherapy visits over 28 days (Mirovsky et al., 2006). Two RCT's using walking interventions to treat pain compared to an eight-week supervised exercise and usual care physiotherapy found a reduction in pain between baseline and follow-up and included a mean of 3.5 face to face usual care physiotherapy visits (Eadie et al., 2013; Hurley et al., 2015). Exercise classes which had a face-to-face interaction, ranged from four (Hurley et al., 2015), and 4.7 (Eadie et al., 2013) to 36 visits in Torstensen et al., (1998), to achieve reduced pain intensity. Telephone contacts used in walking interventions ranged from six (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015) to eight (McDonough et al., 2013). Although, it appears that RCTs with fewer than 36 visits in less than 12 weeks demonstrated reduced pain intensity in those with CLBP (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). None of these studies used number of visits as an independent variable. The current RCT allows for deeper understanding of the dosage per treatment group required for improved outcomes.

Related to the number of physiotherapy visits is the teaching of exercise. Exercises are considered best practice for CLBP (Chou et al., 2007; Chou, 2010; Gordon and Bloxham, 2016). These exercises can vary in duration, frequency and intensity upon the patient's interpretation once done outside of the clinical setting. The current RCT methodology used exercise as a taught modality in all three treatment groups. In a prospective, observational cohort study of 54 outpatient physiotherapy practices in Israel, a higher compliance to active home exercise was associated with better outcomes (Deutscher et al., 2009). Furthermore, concurring evidence states therapeutic alliance may improve patient compliance in home exercises (Deutscher et al., 2009; Hall et al., 2013). Both compliance rate of attending scheduled visits and doing home exercises is lower in lumbar spine problems than in shoulder and knee problems (Deutscher et al., 2009). The authors did not investigate the exercise improving with compliance but suggested that the relationship of the physiotherapist to the patient involved selling or convincing the patient of the importance of physiotherapy and home exercises (Deutscher et al., 2009). A similar finding was seen in a meta-analysis of forty RCT's on CLBP exercises where reductions in CLBP pain intensity were observed with treatment when compared to minimal or no treatment (Ferreira et al., 2010). Of 40 RCT's, only three included unsupervised exercise treatment. The number of exercise sessions was significantly associated with the effect of exercise on pain intensity ($p=0.028$). The study suggested that with each extra exercise session, the effect size would increase by 0.13 on a 100 - point continuous scale measuring pain intensity (95% CI=0.02 to 0.24). Although significantly associated with outcome, the number of sessions only has a small impact on exercise treatment effect since 8 additional exercise sessions would have to be added to provide an extra 1 - point difference on a 100 - point NRS difference between an exercise

intervention and control. The total number of exercise hours was not significantly associated ($p=0.25$) with exercise effect size (Ferreira et al., 2010). This supported the argument for the influence of supervisory relationship influencing pain intensity and not the length of time spent together. Despite the small effect on pain, there appears to be a link between the number of supervisory exercise sessions and reduced pain intensity in CLBP. With all three treatment groups in the current RCT receiving face to face supervision and a form of exercise to be done in treatment and at home, the association with supervision and exercise may explain reduced pain intensities in all three groups.

A systematic review demonstrated that the alliance between therapist and patient appears to have a positive effect on outcomes associated with CLBP (Hall et al., 2013). Although, this alliance was not defined explicitly. The current RCT demonstrates a significant association between number of physiotherapy visits and treatment group ($p<0.01$). Those in the W treatment group had fewer physiotherapy visits than those in the P and PW treatment groups. Despite this, participants in the W treatment group showed small yet improved mean pain intensity score at 12-week follow-up. Therefore, although not receiving a hands-on approach of massage and manipulation combined with exercise, the presence of the physiotherapist to supervise a walking intervention may reduce pain intensity, although differences in pain intensity may not be clinically meaningful in the W treatment group. The physiotherapy visits supervision in all three treatment groups had common factors of supervising exercise (either walking or isometric lumbar stabilization), pain education and advice to stay active. These factors may contribute to the therapeutic alliance and the influence on pain intensity reduction.

The number of physio visits within studies may be determined by factors including, financial resources, practice settings and responses to treatment (Gracey et al., 2002). As indicated by the NICE clinical guidelines, No.88, for Low Back Pain, the guidelines advocate eight sessions when offering a structured exercise program, nine treatments when applying manual therapy, or ten sessions of acupuncture over a 12-week period for non-specific CLBP (National Collaborating Centre for Primary care [UK], 2009). Methodology in the current RCT was in line with UK NICE guidelines recommending between 8-10 physiotherapy visits (National Collaborating Centre for Primary Care [UK], 2009). Patients with CLBP within LBP cohorts were treated more frequently than acute LBP (Swinkels et al., 2005; Groenendijk et al., 2007). Sub-acute and CLBP received 2.3 more visits than acute LBP (Swinkels et al., 2005). Similarly, Groenendijk et al., (2007) found a significant increase in the number of CLBP treatments between 1989/91- 2002/3 when acute LBP treatment number had decreased. This is of interest since RCTs using walking interventions to treat CLBP demonstrate that reducing the number of hands-on treatments may be optional as a treatment (Torstensen et al., 1998; Mirovsky et al., 2006; Eadie et al., 2013; McDonough et al., 2013; Hurley

et al., 2015). Minimum clinically important differences and effect sizes should however be considered when offering walking exercise as a treatment alone.

The number of physiotherapy visits is influenced by a number of organisational infrastructure factors too. A multilevel analysis involving 1733 patients at 41 practices treated by 97 physiotherapists in the Netherlands explained variation in visits was determined by patient, practice, or physiotherapist. The Netherlands study showed that the number of physiotherapy visits depends mainly on patient characteristics ($p<0.001$) (Swinkels et al., 2005). Patients having more physiotherapy visits were older, female, having chronic complaints, having had previous therapy, referred by a specialist (Swinkels et al., 2005). Similarly, participants seen in the current RCT, suffering with CLBP were mostly female, with the entire cohort with a mean age of 46 years. In the current RCT, the number of visits was not compared to these patient level characteristics. With these demographics represented in the RCT, it may have attracted the attention of these patients as the methodology supported three to nine treatments.

Physiotherapist characteristics are known to influence number of physiotherapy visits (Swinkels et al., 2005). Although most of the variance was in patients, therapists with additional training in LBP treated their patients 1.5 sessions fewer times than patients treated by other physiotherapists (Swinkels et al., 2005). Physiotherapists that were female, or older (>45 years) or working less than 20 hours a week, treated patients fewer times (Swinkels et al., 2005). Physiotherapists that were male, worked more than 40 hours a week, or younger than 45 years of age treated patients more frequently (Swinkels et al., 2005). In the current RCT participants were seen by a balance of male and female physiotherapists, although experience treating LBP was not quantified. The RCT methodology sufficiently prepared all the qualified physiotherapists to deliver similar evidence-based care. Physiotherapist level factors appear highlighted with the current evidence that the number of physiotherapy visits is associated with pain outcomes. Factors that influence the number of physiotherapy visits have not been explored in the reviews using walking to treat CLBP (Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

Physiotherapists in the UK and the Netherlands have noticed changes in the number of physiotherapy visits by patients. A cross-sectional voluntary electronic survey was conducted and 223 musculoskeletal physiotherapists in the UK were asked to gauge the relevance and practicality of NICE guidelines for LBP (Parr and May 2014). NHS physiotherapists commented that the numbers of visits recommended in the guidelines were not realistic and that limitations were placed on them, limiting patient's follow-up appointments due to financial constraints represented in current economic policies. Private practitioners in the same paper suggested that medical insurance companies were restricting the number of physiotherapy visits making therapeutic benefit impractical

(Parr and May, 2014). This decrease in visits is echoed in a study of 3148 patients treated for LBP by physiotherapists in the Netherlands aimed to see if there was a change in knowledge and health policy from the period 1989-1992 to 2002-2003 (Groenendijk et al., 2007). This decrease was in acute LBP and not CLBP treatment though. The results show quality management by professional bodies and volume policy by government and insurance companies have decreased the number of visits and increased use of evidence-based interventions for acute LBP. The former period had an average number of 11.3 (S.D. 1.4) visits. A significant decrease of 1.1 visits per treatment episode of each patient was measured in the later period ($p<0.001$). Subgroup analysis showed that this decrease was only in public health insured patients and not private health insurance. It appears that quality management and volume policy in improving treatment efficacy by the Netherlands government and the use of evidence-based interventions have been the driving forces for this change. With the absence of NICE guidelines for the management of CLBP in South Africa, the methodology used in this RCT was implemented to carry out a level of evidence-based care regarding number of visits. In typical South African physiotherapy private practice, patients can receive as many visits as they can afford in their own capacity, as well as the amount individual's medical insurance may cover. This was not the focus of this RCT.

Even though face to face physiotherapy visits were utilized for all three treatment groups in this RCT, it is notable to mention the varied types of visits or physiotherapist interactions physiotherapists had with patients in other studies. In other similar studies on CLBP, physiotherapy visits are typically conducted in person, and are referred to as visits, treatments, or sessions (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015). Four primary studies in these two systematic reviews used telephone calls for physiotherapists to engage with participants and were referred to as contacts (Torstensen, 1998; McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Where walking as a sole intervention has been used to treat CLBP, methodology of physiotherapy visits or participant supervision was observed in varied guises; either as, face to face supervision (Shnayderman and Katz, 2013), telephone contacts (Torstensen et al., 1998; McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015) or logging onto the internet in an internet-based treatment (Krein et al., 2013). The effect of different interactions with patients may be unknown and the advantage of the current RCT was that patient interaction was consistent using face to face supervision in all three treatment groups. This was not consistent in related literature (Torstensen, 1998; McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015).

Related to the number of visits may be a placebo response. Information indicates that increased visits to health care practitioners improves results (Ilnyckyj et al., 1997; Deutscher et al., 2009). Contrary to this, a meta-analysis of placebo response in irritable bowel syndrome trials showed lower placebo response with increased visits (Patel et al., 2005). The same meta-analysis did suggest that this lower

placebo response may have been due to three factors. Factors included; investigator interaction opposed to clinician interaction; increased clinician visits may introduce inadequate blinding and may lead patients to suspect they were receiving placebo causing drop out, and dissatisfaction when insufficient pain relief was obtained (hence the addition of extra visits) (Patel et al., 2005). Previously shown, a strong doctor-patient relationship improves clinical outcomes (Patel et al., 2005; Tavel, 2014). Highlighted were warmth and empathy, both contributors to placebo (Patel et al., 2005; Tavel, 2014). These were not measured in the current RCT, nor in studies using walking and physiotherapy to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). However, these factors, for example, may have contributed to participating physiotherapists being hired to work in private practice.

Understanding concepts within pain neuroscience may be a salient point when examining pain intensity as an outcome. Two reviews focussed on the neurocognitive aspects of pain perception using placebo induced analgesia as an example, and mention that mechanisms of expectation, attention, and reappraisal in pain cognitive modulation (Wiech et al., 2008; Bystad et al., 2015). Neurocognitive pain modulation refers to modulation of pain through several brain areas, neural pathways, and brain cortical networks. It seems likely that neurocognitive pain modulation may be an important component in pain intensity reduction in this RCT as well as, however unmentioned, in reviews where walking and physiotherapy was used to treat CLBP pain intensity (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). Recent advances in neuroscience show neurocognitive pain modulation evidence using fMRI and describe it as the dynamic pain connectome (Kucyi and Davis et al., 2015). The evidence describes interactions of three key brain systems involved in spontaneous attentional fluctuations toward and away from pain: the salience network, the default mode network, and the antinociceptive system. The clinical interpretation may be that there are potentially people who are more prone to attention to pain, and those who are not (Wiech et al., 2010; Kucyi and Davis et al., 2015). The paper demonstrates the effect of attention modulation away from the pain. This RCT's treatment groups may have shown improved outcomes due to attention to non-painful stimulation or distraction which is known experimentally to decrease pain intensity and attenuate nociceptive brain neuronal responses (Eccleston and Crombez, 1999; Hayes et al., 1999; Iwata et al., 2005; Morrison et al., 2013). Mindfulness meditation training, meditation, and cognitive-behavioural therapy are said to engage the mind in mind wandering away from the pain in a non-effortful manner (Kucyi and Davis et al., 2015). Similarly, the treatments used in this study with individual components and/or the common components of pain education and exercise (walking, isometric lumbar stabilization, or a combination) may enhance attention modulation away from the pain.

A review on placebo described supportive patient-practitioner components of the placebo effect and that the effect is enlarged by hands-on contact with close verbal communication between therapist and patient (Tavel, 2014). This was the design of two of the groups (P and PW) in this RCT. The W group in the current RCT also showed small mean reductions in pain, implicating the hypothesis that close verbal communication may be influential in pain reduction. However, the results of the current RCT showing a MCID in pain reduction at 12-weeks in only the PW group concurs with the evidence that the placebo effect is enlarged by hands-on contact with close verbal communication between therapist and patient. An earlier review expands on related objective measurement of fMRI studies in pain analgesia exploring the association of therapeutic relationships and reduced pain outcomes (Benedetti et al., 2005). This is furthermore corroborated in a RCT of 262 patients with irritable bowel syndrome (Kaptchuck et al., 2008). A waiting list, (observation), was compared to placebo acupuncture alone, and placebo acupuncture with patient practitioner relationship augmented by warmth, attention, and confidence. The key finding was that placebo plus empathy/warmth was more effective than placebo alone. Findings included global improvement, relief of symptoms, improved symptom severity and improved quality of life. Demonstrated components (assessment and observation, a therapeutic ritual, and a supportive patient-practitioner relationship) could be progressively combined resembling a graded dose escalation of component parts with the patient practitioner relationship having the greatest effect (Kaptchuck et al., 2008). The relationship was specified as comprising of individual factors of warmth, duration of interaction, empathy, and communication of a positive expectation (Kaptchuck et al., 2008). A systematic review listed 67 communication factors correlating with the therapeutic alliance, and implicated patient centred communication to be the focus to strengthen this alliance (Pinto et al., 2012). The concepts of placebo, therapeutic alliance, warmth, and empathy appear to be associated with reducing pain symptoms and may be important considerations when treating pain. This was not discussed in the reviews using walking and physiotherapy to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018).

It is unknown if the significant association with pain intensity outcome at 12-week follow-up would continue with more physiotherapy visits. Unlike the current RCT, a study conducted in Israel found a high number of visits for LBP were associated with poorer outcomes including pain intensity (Deutscher et al., 2009). The current RCT was conducted in a private practice setting unlike the public health care system in Deutscher et al., (2009). In context of the Maccabi public health care system in Israel, therapist reimbursement is not dependant on the number of visits (Deutscher et al., 2009). Their data suggests that when the patient improves, the treatment number is shortened and when patients do not show improvements, the therapists add visits hoping it will improve outcomes. The number of physiotherapy visits highlights the context, content, and organizational infrastructure of health.

Factors involved in treatment

Treatment in this RCT were based on standardized modalities recommended as part of UK NICE guidelines for usual care physiotherapy (National Collaborating Centre for Primary Care [UK], 2009). Guidelines proposed by the ACSM were used for the walking intervention. Results in this RCT corroborated findings in previous walking intervention RCTs with no statistically significant difference in pain outcome measures between treatment groups (Eadie et al., 2013; Hurley et al., 2015). These previous studies using treatments sanctioned by NICE and ACSM, together with the current RCT showed reduced pain intensity in all three treatment groups. Aside from the placebo effect or regression to the mean, several factors are worth mentioning regarding treatments in physiotherapy and walking trials.

Varied modalities can be applied by physiotherapists, and all may have an effect on pain. The combination of physiotherapy treatments and modalities for treating CLBP vary due to physiotherapist's discretion as seen in previous trials (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015). However, three core modalities of manipulation, massage and lumbar stabilization exercise utilized in unknown amounts are described in the literature as part of usual care physiotherapy (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015). The current RCT delivered these modalities as physiotherapy usual care with no discretion on type, and amount of modality used but delivered in a standard manner. Regardless of which discretionary combination of modalities were used in usual care physiotherapy groups (ice, heat, massage, manipulation, electrotherapy, exercise), pain intensity was also reduced in previous usual care interventions (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015).

Used alone or in combination, objective PA monitoring, education on CLBP, and exercise, may improve pain and associated outcome measures. An American RCT compared a pedometer-based walking program with motivation, feedback and social support to the comparative arm which received no support but only a pedometer with which to measure steps taken (Krein et al., 2013). Both treatment arms utilized a pedometer and therefore were potentially motivational to increasing participant steps (Krein et al., 2013). Without the educational component, exercise, or walking intervention the comparator arm also achieved small reductions in pain. The pedometer may have affected the participants feeling of care. Walking interventions may show benefit if they are objectively monitored (using a pedometer), graded via an evidence base, and are combined with pain education. This was observed in the current RCT. However, albeit showing a reduction in pain intensity, PA was unrecorded using a pedometer in intervention arms of usual care physiotherapy, and supervised exercise (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015). Additionally, explicit pain education on CLBP was absent in one of these RCTs (Torstensen et al., 1998). The

combinations of these modalities are suggestive of other factors involved in treatment effect on reducing pain intensity.

More recently, standardized evidence-based forms of education on CLBP are frequently now referred to as PNE, are used to reduce pain intensity (Puentedura and Flynn, 2016; Hush et al., 2018). Three previous RCT's compared a usual care physiotherapy intervention to an exercise intervention including strength and flexibility exercises and a walking intervention for CLBP (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015). Of these, only two RCTs delivered explicit advice and education using 'the Back Book' to participants (Eadie et al., 2013; Hurley et al., 2015). The Back Book is intended as a valid and current guide for patients and clinicians containing advice based on the evidence-based research for management of LBP and mitigating disability (Roland, 2002). In three RCTs using walking as a CLBP treatment however, physiotherapists gave individualized education/ advice and the patients received information from 'The Back Book' promoting self-management (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). All intervention groups using 'The Back Book' showed reduced pain intensity at follow-up. The intervention groups included usual care physiotherapy, strength, and flexibility exercise, walking programs or PNE. Unlike these studies using the Back Book, the current RCT did not use standardized information but did educate patients on CLBP. Each physiotherapist was tutored in the understanding of CLBP according to NICE guidelines (National Collaborating Centre for Primary Care [UK], 2009). Each of the modalities applied, alone or in combination were used internationally as part of evidence-based practice. Physiotherapists were instructed to encourage participants with the knowledge of PNE focussing on the neurobiology of pain (Moseley, 2003; Moseley and Arntz, 2007; Louw et al., 2011; Puentedura and Louw, 2012; Butler and Moseley, 2013). The method used was to educate patients about neurophysiological processes involved in pain experiences rather than focussing on tissue pathology. The common factor between the previous studies using education and the current RCT was that patients received a form of education proposing management of CLBP. This interaction may have been responsible for reduction in pain intensity. The explicit absence of PNE may explain why some study interventions demonstrate significant differences in pain intensity. In some CLBP intervention studies, patient education on the self-management of CLBP was not described as being included in any of the treatment groups (Torstensen et al., 1998; Mirovsky et al., 2006). During manual therapy or exercise interventions, physiotherapists are face to face with participants (Torstensen et al., 1998; Mirovsky et al., 2006). During face-to-face therapy there may be delivery of positive advice on CLBP, not explicitly PNE, which may positively assist the patient in behaviour change and self-management. Manual therapy was shown to be efficacious in treating pain when compared to walking and exercise interventions in a RCT comparing usual care physiotherapy to stretching and strengthening exercises or walking as self-exercise (Torstensen et al., 1998). This was controversial as heat, massage, TENS, and traction had been shown no better or no worse than a

placebo or control group in comparable physiotherapy studies (Van der Heijden et al., 1988; Manniche et al., 1988). The authors did not elaborate whether physiotherapists delivered pain education and if so, what kind of advice or education was included (Torstensen et al., 1998). Up to now, it appears that delivery of explicit reproducible education about CLBP may be an important factor in reducing pain intensity.

Similarly, exercise in combination with education on CLBP may reduce pain. The comparative intervention arms using physiotherapy usual care and exercise therapy all used standardized education via 'The Back Book' (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). A internet-based walking intervention had internet-based educational materials only for the walking intervention (Krein et al., 2013). All four trials using exercise combined with education showed small yet reduced mean pain scores (McDonough et al., 2013; Eadie et al., 2013; Krein et al., 2013; Hurley et al., 2015). Concurring with these results, exercise was combined with education on CLBP in the current RCT. In a trial that examined the impact of walking as self-exercise for CLBP treatment where no pain education was received, found that those who walked compared to stretching and strengthening exercise or usual care physiotherapy did not show significant pain reduction (Torstensen et al., 1998). Education on how to manage one's CLBP pain symptoms may be a fundamental factor in reducing pain regardless of treatment modalities.

For more than a decade a trend has been to move patients away from passive treatments such as massage and manipulation, towards prescribing exercise, education, and self-management for treatment of pain in those that have CLBP (Moseley, 2007; Groenendijk et al., 2007, Dean and Duncan, 2016). A concessionary view was conveyed, however, in a recent review of 13 papers showing LBP outcomes favoured a combination of manual therapy and/ or an exercise-based strategy in combination with PNE when compared to outcomes of education only approaches (Louw et al., 2016). This elucidates the possible need for manual therapy in context, with appropriate education and advice delivered during physiotherapy visits. This evidence concurs with results seen in the current RCT and those using interventions of manual therapy, strength and stretching exercises or walking combined with PNE (McDonough et al., 2013; Eadie et al., 2013; Krein et al., 2013; Hurley et al., 2015). Furthermore, pain related outcomes were further improved when walking was combined with PNE when compared to PNE alone (McDonough et al., 2013).

Aside from education on CLBP, varied forms of exercise, specifically when supervised are shown to improve CLBP pain outcomes, noted in a systematic review and meta-analysis (Hayden et al., 2005a; Ferreira et al., 2010). Both isometric lumbar stabilization and walking were used as exercise interventions in this RCT. The isometric lumbar stabilization taught may have had a carry-over effect when done at home as seen in Ferreira et al., (2010). Both P and PW interventions in the current RCT

used this modality. Concurring with this were interventions in previous walking intervention studies in the usual care physiotherapy intervention arm, which showed pain reduction outcomes (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015).

Exercise received by participants from a clinician differs in nomenclature, and is referred to as prescribed, dose or load, and pacing depends on the individual (Hayden et al., 2005a; American College of Sports Medicine, 2011). Graded frameworks of exercise graded by dose or pacing in walking may improve pain intensity outcomes. These varied factors may influence the mechanism of pain reduction. The mechanisms that may be responsible for pain intensity reduction through exercise are not clearly stated in previous reviews using walking and physiotherapy led lumbar stabilization exercises (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al 2018). The isometric lumbar stabilization exercise effect may decrease pain globally through the hypothesis that muscular hypertrophy and spinal stiffness are beneficial to the spine (Stanton and Kawchuk, 2008; Katsura et al., 2011). In the walking intervention, the assumption is made in Vogt et al., (2003) that altered hip extensor recruitment patterns alter muscle activation patterns impacting on the physiological loading and alter the direction and magnitude of joint reaction forces. Previous CLBP exercise studies emphasize biomechanical reasons why muscles involved in walking may reduce pain through muscular strength and neuromuscular control (Stanton and Kawchuk, 2008; Katsura et al., 2011; Vogt et al., 2003). Two physiotherapy reviews suggest aerobic exercise modulates CLBP by increasing blood flow and nutrients to lower back soft tissues, augmenting healing, and decreasing stiffness (Gosling, 2013; Gordon and Bloxham, 2016). The effect of exercise mechanisms on pain reduction remains multi-factorial.

This RCT demonstrates that walking may have clinical application as an intervention for CLBP if combined with usual care physiotherapy. However, as there was no statistically significant difference between treatment groups in pain intensity, it remains unclear whether physiotherapist supervised exercise combination of walking and/or isometric lumbar stabilization exercise, or the beneficial effects of manual therapy, the placebo effect, or a combination of these factors lead to decreased pain intensities. None of the studies using walking as a CLBP treatment have been able to elaborate fully on the mechanisms involved in reducing pain intensity (Mirovsky et al., 2006; Koldaş Doğan et al 2008; Eadie et al., 2013; McDonough et al., 2013; Karadeniz et al., 2014; Hurley et al., 2015 Magalhães et al 2015; Cho et al., 2015; Lee and Kang 2016).

The perception of any treatment as beneficial may play an important role in reducing pain related outcomes. The ‘Hawthorne effect’ is described as the alteration of behaviour by the participants of a study due to their awareness of being observed (Fry, 2018). Factors associated with the ‘Hawthorne effect’ including physiotherapy visits, completion of repeated outcome measures, using pain and

activity diaries, and wearing of pedometers may be of influence in pain reduction in all three groups. The Hawthorne effect is seen in improved outcomes across trials regardless of explicit treatments in the current RCT or discretionary use of modalities in previous studies.

Expectation of pain following treatment

From previous studies, patient expectation of the resolving of their pain has closely been studied alongside satisfaction, being similar but not the same (Bleich et al., 2009; Forero and Gómez, 2017). It is noted that satisfaction with treatment for reducing pain symptoms is associated with a 'post purchase' perception whereas expectation related to outcome is more related to a 'pre-purchase' perception (Forero and Gómez, 2017). The ontology of these two categories differs, where satisfaction is related to a retrospective view and expectation may be associated to a prospective view. In this RCT, expectation of pain outcomes was assessed in the entire cohort at baseline. A positive correlation was noticed between expectation of pain reduction and actual reduction in the whole cohort. Participants who expected less pain at 12-week follow-up demonstrated lower pain intensity at the 12-week follow-up. Expectation can influence outcome in terms of pain and has been corroborated and discussed elsewhere (Wiech et al., 2008; Atlas and Wager, 2012; Campbell et al., 2013).

Neurocognitive mechanisms of expectation may result in pain modulation (Wiech et al., 2008; Bystad et al., 2015). This may explain the result achieved in all three treatment groups. Expectation is a learned response involving previous positive experiences with a therapy, augmenting the placebo literature (Wiech et al., 2008; Colloca and Miller, 2011; Reicherts et al., 2016). Participants who had received previous physiotherapy were not excluded from this study, and so it is possible that they had had positive previous physiotherapy experiences. It is unclear if these associated factors were learned prior to entering the trial or developed after the trial began.

It is not clear whether expectation or patient education in the initial appointment and assessment, the number of physiotherapy visits, exercises completed, or a combination of positive expectation, education and therapy received explained the decrease seen in pain intensity in the three treatment groups.

Nociceptive pain and neuropathic pain

Using the painDETECT questionnaire to determine pain phenotype, in this RCT, demonstrated that the clinical burden was greater for those patients with neuropathic CLBP compared to those with nociceptive CLBP across the entire cohort. This concurs with other studies (Chetty et al., 2012; Smart et al., 2012b; Baron et al., 2016; Spahr et al., 2017). It is not known what the clinical burden of pain phenotypes was in previous studies using walking to treat CLBP as pain was treated as a homogenous

entity (Torstensen et al., 1998; Mirovsky et al., 2006; Koldaş Doğan et al 2008; Eadie et al., 2013; Krein et al., 2013; McDonough et al., 2013; Hurley et al., 2015; Magalhães et al 2015; Cho et al., 2015; Lee and Kang, 2016).

The NeuPSIG guidelines on CLBP and PNP assessment in daily practice and clinical trials recommend measuring pain (Haanpää et al., 2010). Clinical practice research demonstrates the need for further neurophysiological understanding in in the treatment of CLBP (Moseley, 2007; Naidoo et al., 2012). Specifically, it is noted that South African physiotherapists have a poor understanding of the neurophysiology of pain (Naidoo et al., 2012). Hence, the current RCT may have clinical relevance for physiotherapists.

The insight that pain related outcome measures in this RCT may present with greater patient burden for the neuropathic pain phenotype, may aid patient treatment through education and understanding of different outcome presentations when treating patients with CLBP. This RCT was not powered for six treatment groups (both pain phenotypes for each treatment group), but only for three treatment groups. Previously TENS and "specialised physiotherapy" had been mentioned as possible treatments for neuropathic pain (Chetty et al., 2012). This RCT does not demonstrate intervention effect on pain phenotype only greater post treatment burden with pain and disability outcomes remaining in the neuropathic pain phenotype.

Further investigation into clinical management to improve neuropathic pain outcomes has been called for (Chetty et al., 2012). Patients can be informed of possible differences between pain phenotype presentations when discussing pain management.

Effect of pain on dropout

Walking interventions have been observed to reduce CLBP pain intensity and have had good adherence to walking protocols (McDonough et al., 2013; Krein et al., 2013; and Hurley et al., 2015). In this RCT, the largest drop out was found in the W treatment group. However, those in the W treatment group who were able to persist with the RCT were able to achieve a statistically significant reduction in pain intensity but not a clinically significant reduction in pain intensity. The patients that remained in the RCT, specifically in the W treatment group may have been a select group which did not avoid PA, indicating bias in the sample. Pain persistence or avoidance scenarios are previously documented. A study of 79 patients with CLBP were classified according to scores on the Patterns of Activity Measure-Pain into persisters, avoiders, mixed performers (high persistence and avoidance scores) or functional performers (low scores on both persistence and avoidance behaviour) (Huijnen et al., 2011). Measured using an accelerometer, the objectively measured PA over 14 days did not differ between groups (Huijnen et al., 2011). Since these differences in activity related behaviour

were not measured in the current RCT, further investigation may be warranted. To manage some of the potential bias, last observed pain intensity for the drop out analysis was analysed.

There was a significant effect of pain intensity at baseline (p=0.01), and last observed pain intensity score (p=0.01) on drop out, in the W treatment group compared to the PW treatment group. Greater scores of pain intensity at baseline, and high last observed pain intensity scores predicted drop out. Since pain intensity and pain phenotype were confounding variables, either may be responsible for dropout. A fisher's exact test demonstrated no association between pain phenotype and drop out (p=0.09). However, when examining the effect of baseline pain intensity on dropout, the odds of dropout were higher for the neuropathic pain phenotype compared to the nociceptive pain phenotype (OR 2.55 (95% CI 1.04 to 6.22)). Although when analysing the effect of last observed pain intensity, it appears that participants who had neuropathic pain phenotype would remain in the RCT since no significant effect on pain phenotype was shown in the data. If pain intensity was high as the RCT progressed, patients were likely to dropout.

When treating CLBP patients using walking as an intervention for reducing pain intensity, careful clinical choice in deciding whether a walking program will be appropriate for patients may be necessary to avoid treatment termination. This opinion is supported early in the RCT where five participants who were randomised to the W treatment group discontinued the treatment because they were expecting manual therapy in usual care as offered in P and PW treatment groups. Other walking interventions have used the Self-efficacy scale measures to examine the extent to which participants could master their ability to persist with exercise and/ or walking in varied situations (McDonough et al., 2013; Hurley et al., 2015). Self-efficacy improved in the walking and education as well as the education only group, and participants in the walking group had 70% adherence to their weekly step targets (McDonough et al., 2013). Other trials however postulated that participants underestimate the difficulty of changing their exercise behaviour at baseline (Krein et al., 2013; Hurley et al., 2015). Both authors reported reduced self-efficacy post RCT and suggested future investigations, as their data appeared counter intuitive (Krein et al., 2013; Hurley et al., 2015). In order to analyse participants coping strategies with either persisting or avoiding further activity, the current RCT did not use a Self-Efficacy scale, Chronic Pain Coping Inventory or Patterns of Activity Measure-Pain scale (Jensen et al., 1995; Huijnen et al., 2011). In the current RCT, patients in the PW treatment group were more likely to remain in the RCT compared to W and P treatment groups. Related factors were not measured; however, the combination of therapies may have been more motivating and encouraging to participants. Clinically, the results may encourage the use of a pedometer-based walking program with usual care physiotherapy in patients with CLBP.

Disability

A recent systematic review using walking as a treatment for CLBP concentrated on disability as the primary outcome (Lawford, Walters and Ferrar, 2015). It stated that walking was as effective as usual care, strength specific exercise, medical exercise therapy and supervised exercise classes to improve disability outcomes in adults with CLBP. Although the current trial did not concentrate on treatment effectiveness on disability, the results demonstrate no statistically significant between-groups differences in ODI scores between baseline and 12-week follow-up were seen. For the first time, P, PW, and W treatment groups were compared to one another including ODI as an outcome. However, even though all three treatment groups reported improved mean ODI score from baseline to 12-week follow-up, the greatest improvement in mean disability scores were observed in the PW treatment group. These results concur with latter systematic review furthermore recommending the use of walking and physiotherapy treatment for CLBP (Sitthipornvorakul et al., 2018). It is unclear at this point which factors involved in these treatments are more efficient to treat disability in CLBP.

The Institute for Health Metrics and Evaluation (IHME) (2019) published their findings in the WHO publication 'Global Burden of Disease', headlining LBP, which included CLBP, as a leading cause of disability. Disability from LBP is greater than that caused by headache disorders, depressive disorders and diabetes (IHME, 2019). In the current RCT, disability measured with the ODI was significantly associated with pain intensity at 12-week follow-up, indicative of the relationship of disability and pain intensity in the clinical scenario. However, pain is not always an indicator of tissue damage (Eccleston and Crombez, 1999). The causes of disability can have numerous factors (O'Sullivan, 2005; Di Iorio et al., 2007; Chou, 2010; Schaller et al., 2015). Some of these are discussed below.

The analysis demonstrated the effect of increased age, and unemployment on ODI score at 12-week follow-up. In the entire cohort at baseline, the mean participant age was 46.2 years, and 8.2% of participants were unemployed. The effect of age was significant, controlling for the other variables in the model. In the current RCT, age remained associated with ODI score, which concurs with studies on Japanese and Italian CLBP populations (Tonosu et al., 2012; Di Iorio et al., 2007). The current RCT ODI score was greater for those unemployed compared to those employed ($p=0.03$) at 12-week follow-up. This narrative is reflected in a review stating that return to work be an essential outcome for chronic pain patients to reduce associated disability (Sullivan and Hyman, 2014). The association highlights that people who have greater disability from CLBP may be off work. At baseline, the current RCT cohort demonstrated moderate disability and included mostly employed patients (91.8%). This could be interpreted that moderate disability may not be sufficient to prevent work in many people, or that this cohort persisted in work despite associated disability.

Results from CLBP studies using education on CLBP, walking, strength and flexibility exercise classes, and usual care physiotherapy, demonstrate the possibility that one or many factors involved in these treatments may reduce disability (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Varied education seen in pain management programs, typically focussing on cognitive and behavioural aspects, are constructive in reducing disability, normalising pain cognitions and promote self-efficacy (Moseley, 2002). Two reviews of physiotherapy pain management suggest exercise related factors globally reduce disability from CLBP (Gosling, 2013; Smith et al., 2014). Factors mentioned are muscle contractions mobilizing chemical irritants, non-opioid activation through descending inhibition via central nervous system effects together with positive psychophysiological adaptations (Gosling, 2013). Furthermore, improved musculature dampening forces may improve disability associated with CLBP (Smith et al., 2014). Pedometer-based walking and isometric lumbar stabilizations used are both considered as exercise. Usual care physiotherapy including massage and manipulation may reduce pain and furthermore encourage functional movement, reducing disability (Moseley, 2002).

In studies of physiotherapy treatments including patients with CLBP, disability outcome measure scores appear to be reduced if exposed to increased number of treatments (Deutscher et al., 2009; Ferreira et al., 2010). In the current RCT, pain intensity at 12-weeks was associated with number of physiotherapy visits, so the number of physiotherapy visits was excluded from the analysis of ODI score. Although physiotherapy visits were excluded, the factor that was shared between all three treatment groups was exercise. Isometric lumbar stabilization, walking and the combination of both were exercise modalities in all three treatment groups in the current RCT which may have contributed to the effect of reduced ODI scores at 12-week follow-up. This concurs with the findings in two RCTs which showed no statistically significant difference between groups in disability scores, all interventions included exercise and showed reduced mean disability (Eadie et al., 2013; Hurley et al., 2015). As with pain intensity outcomes, a meta-analysis showed high doses of exercise were shown to reduce disability from CLBP (Hayden et al., 2005b). Previous CLBP epidemiological studies, and systematic reviews on CLBP treatment concur that CLBP is the leading cause of limiting activity (Chou, 2010; Schwellnus, 2011; Schaller et al., 2015). Hence, the importance of exercise associated interventions has been a focal point in CLBP research.

Walking as exercise is evidenced to influence fitness, strength, and mobility of tissues of patients, possibly contributing to reduced disability from CLBP (Lawford, Walters and Ferrar, 2015). Disability may be influenced using a walking intervention if methodology of its application is evidence based and/or monitored objectively using graded doses of exercise. The current RCT and three others studied disability when comparing usual care physiotherapy to both exercise and walking interventions (Torstensen et al., 1998; Eadie et al., 2013; Hurley et al., 2015). A Norwegian study

found statistically significantly reduced ODI scores in the usual care physiotherapy and exercise interventions groups and not in a self-paced walking intervention (Torstensen et al., 1998). However, the current RCT, and two similar RCTs found that ODI score decreased in all groups whether it be usual care physiotherapy, group exercise or a graded dose walking intervention based on ACSM guidelines (Eadie et al., 2013; Hurley et al., 2015). Unlike the Norwegian study, disability scores were reduced in interventions describing graded doses of walking based on evidence-based structure (McDonough et al., 2013; Krein et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Additionally, in one RCT, two groups of walking interventions both graded their dose of exercise over time (over ground compared to treadmill), both showing statistically significant reductions in ODI scores (Karadeniz et al., 2014). In this instance both groups were objectively measured (using heart rate) and combined with usual care physiotherapy (Karadeniz et al., 2014). Mean ODI score differences were not mentioned. In the current trial, the mean changes in ODI score from baseline to follow-up were largest in the group combining walking exercise and usual care physiotherapy. The current RCT is the first trial to compare this combination to its constituent parts and demonstrate the mean ODI score changes in in the PW treatment group.

Furthermore, the current RCT and four other RCTs had participants wearing devices to measure activity in walking interventions (McDonough et al., 2013; Krein et al., 2013; Eadie et al., 2013; Hurley et al., 2015). The RCT conducted in Norway did not have any objective measure of activity grading (Torstensen et al., 1998). A pedometer-based walking program would allow the patient to recognize their levels of activity which may have influenced their activity and disability perception (Baker at al., 2008). It is unknown whether using an objective measure of walking alone reduces perception of disability when compared to other physiotherapy treatments.

A meta-analysis showed high exercise doses (≥20 hours), individualized treatment, supervision of home exercise, and usual care physiotherapy which had an exercise component were associated with reductions in disability (Hayden et al., 2005b). This multiple-approach design applied to one RCT comparing usual care physiotherapy and a treadmill walking intervention, to a usual care physiotherapy and an over-ground walking intervention (Karadeniz et al., 2014). Both interventions used heart rate monitors to objectively measure exercise, had partial supervision, and both showed reduced disability (Karadeniz et al., 2014). Only the group which used over ground walking had statistically significant improvements in ODI scores after the Bonferroni correction. Using over ground walking in the current RCT (PW), the greatest ODI score reduction between three treatment groups was noted at 12-week follow-up. The benefit on disability may be linked to treatment setting (outdoors), a graded dose of evidence-based exercise, an objective measuring device regardless of type, and some level of supervision.

In the current RCT, the usual care physiotherapy group also showed reduced disability however displayed unchanged step counts. The P treatment groups though still measured weekly steps, completed isometric lumbar stabilization exercise, and had a goal to reduce disability scores. Measuring activity, coupled with a set goal may add motivation to improve functional outcomes such as the ODI. However, this objective measure of steps was not seen in usual care physiotherapy which showed improvements in disability and no statistically significant difference between treatment groups in two former studies comparing walking, exercise and usual care interventions (Eadie et al., 2013; Hurley et al., 2015).

Isometric lumbar stabilization was used as an exercise in the P and PW treatment groups which could be the reason that reduced disability was seen in these two treatment groups. This modality of exercise was used in strength and flexibility exercises interventions which also showed reduced ODI scores in former studies (Eadie et al., 2013; Hurley et al., 2015). A systematic review on isometric lumbar stabilization which included 2,359 patients demonstrated statistically significant benefit on disability from CLBP (Smith et al., 2014). One mechanism that may explain why stabilization exercises may improve disability is its function to absorb stressful forces acting on the lumbar spine, increasing spinal stiffness and muscular hypertrophy, described in the motor control model (Richardson, Hodges and Hides, 2004; Behm et al., 2010; Byström et al., 2013). Additionally, if exercise is added to usual care physiotherapy, reductions in disability are expected (Hayden et al., 2005b). In accordance with the evidence on exercise through isometric lumbar stabilization, future patients may use these modalities to minimize their disability. Exercises which were taught during physiotherapy sessions may have been done independently as per the current RCT methodology and are known to reduce disability (Ferreira et al., 2010).

Systematic reviews demonstrate manual therapy used in usual care physiotherapy can also reduce disability from CLBP (Hayden et al., 2005a). The effects of pain-relieving modalities such as massage and manipulation may ease suffering which is known to predict reduced disability (Koes et al., 2010, Vigotsky and Bruhns, 2015; Pereira et al., 2017). This may emphasize the association of pain and disability in clinical practice (Moseley, 2002; Pereira et al., 2017). Therefore, usual care physiotherapy modalities that influence the periphery, mechanics, behaviour, and psychology of a patient might have disability reducing effects.

Disability was examined in the current RCT in the combination treatment (PW) and in one RCT where walking interventions and usual care physiotherapy were combined (Karadeniz et al., 2014). Both RCTs found statistically significant improvements in ODI measures at follow-up. Both RCTs utilized massage in their physiotherapy in combination with walking. This concurs with evidence

that massage with remedial exercises is better than massage alone (Airaksinen et al., 2006). Furthermore, a Cochrane review suggested benefits of massage include improved functional status, especially if combined with exercise and education (Furlan et al., 2002). The current RCT and two other RCTs show no statistically significant difference between walking exercise and usual care physiotherapy including massage, mobilization, and exercises (Eadie et al., 2013; Hurley et al., 2015). However, the current RCT demonstrates a greater mean change in ODI score in the PW treatment group. Despite no statistically significant difference, this is supported with evidence stating massage with remedial exercises is better than remedial exercise alone (Airaksinen et al, 2006).

Due to the association of pain phenotype and disability seen in this RCT, it is unknown if the burden of disability is due to pain phenotype, disability, or pain intensity. This association has been demonstrated in previous detailed studies on pain phenotypes (Shaygan et al., 2013; Spahr et al., 2017; Baron et al., 2016). Neuropathic pain phenotype is predictive of greater disability and is corroborated previously (Spahr et al., 2017; Baron et al., 2016).

Kinesiophobia

The TSK scores at baseline for all three treatment groups in this RCT suggested a kinesiophobic cohort. The TSK cut-off score of ≥37 indicates kinesiophobic behaviour (Vlaeyen et al., 1995; Nicholas et al., 2012). This fear avoidance behaviour was seen in all the intervention groups at baseline of three RCTs using walking interventions to treat CLBP (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). This suggests the importance of fear avoidance and kinesiophobia as outcome measures with CLBP. A RCT (n=52) compared moderately intense treadmill walking to specific lower back exercises, mention a possible selection bias with participants only volunteering who had a low fear of activity (Shnayderman and Katz-Leurer, 2013). The strength of the current RCT was that the effects of the treatment groups used were observable since it was possible to recruit a cohort with high levels of kinesiophobia.

Fear avoidance behaviour can impact on the perpetuation of CLBP related disability through hypervigilance and avoidance behaviours (Martinez-Calderon et al., 2020). The fear avoidance behaviour questionnaire was developed by Waddell et al., (1993). It was used in several studies using walking to treat CLBP (Shnayderman and Katz-Leurer, 2013; Eadie et al., 2013; Krein et al., 2013; McDonough et al., 2013; Hurley et al., 2015). However, in the current RCT the Tampa Scale of Kinesiophobia was used (Miller, Kori and Todd, 1991). Fear of pain in CLBP is broadly defined, however both scales are valid in underpinning the maladaptive psychology observed in CLBP (Martinez-Calderon et al., 2020). The fear avoidance model hypothesises that PA is avoided by some people with chronic pain believing that their pain will increase with increased PA (Leeuw et al., 2007). Fear avoidance behaviour may contribute to increased disability since it contributes to

decreased PA levels (Verbunt et al., 2001; Ryan et al., 2009; Martinez-Calderon et al., 2020). Seeing as this cohort was classified as insufficiently active at baseline, the fear avoidance model may be reflected in this cohort.

In the current RCT there was no statistically significant difference in TSK scores between baseline and 12-week follow-up between treatment groups. In RCTs where a walking intervention is compared to CLBP pain education (McDonough et al., 2013), pedometer wearing with no active or passive treatment exposure (Krein et al., 2013) or to usual care or exercise (Eadie et al., 2013; Hurley et al., 2015), the findings also found no statistically significant difference in fear avoidance between interventions following treatment.

In the current RCT, all three treatment groups had reductions in mean TSK scores at the 12-week follow-up. This concurs with other RCTs using treadmill walking or exercise (Shnayderman and Katz-Leurer, 2013), walking and pain education or pain education (McDonough et al., 2013), and evidence based walking or usual care or exercise (Eadie et al., 2013; Hurley et al., 2015). These studies found no statistically significant differences in fear avoidance between groups at follow-up, although they did detect within-group fear avoidance score improvements between baseline and follow-up. The current RCT results demonstrate the PW treatment group presented with the greatest mean changes in TSK scores in the three treatment groups at 12-week follow-up. Only one trial in the three reviews in Table 19 analysed this combination of walking exercise and usual care physiotherapy (Karadeniz et al., 2014). No mean changes in TSK scores were analysed in that trial. The evidence from the current RCT highlight the requirement to examine CLBP biopsychosocially. Two recent systematic reviews on using walking as an intervention for CLBP included the studies using fear avoidance behaviour outcomes (Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). Fear-avoidance outcome measures were not used in studies in an earlier review using walking to treat CLBP and LBP (Hendrick et al., 2010). This may demonstrate the movement towards a biopsychosocial approach of CLBP over time. Kinesiophobia was developed as an outcome for chronic pain in 1991 (Miller, Kori and Todd, 1991). Therefore, this measure for CLBP treatment seems appropriate with walking interventions in the current RCT and in recent reviews using fear avoidance measures (Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018).

Physiotherapy treatment involving many modalities (e.g.: exercise, mobilization) is now established as treatment for kinesiophobia (Martinez-Calderon et al., 2020). Isometric lumbar stabilization was one exercise used in the current RCT, and interventions in RCTs also using walking to treat CLBP (Eadie et al., 2013; Hurley et al., 2015). Due to improved scores in fear avoidance and kinesiophobia over time in all the treatment groups, the current RCT concurs with previous RCTs, that isometric lumbar stabilization may have a positive effect on reducing kinesiophobia (Eadie et al., 2013; Hurley

et al., 2015). It too has been shown to be effective in treating CLBP pain and disability outcomes (Smith et al., 2014). However, it has been criticized for increasing fear avoidance behaviour, promoting unhealthy beliefs and thoughts on pain and function in other studies (Unsgaard-Tøndel, 2010; Marshall et al., 2013; Nijs et al., 2013). When comparing treatments, a trend towards fear avoidance in stabilization exercises compared to stationary bikes, slings, and general exercises is noted (Unsgaard-Tøndel, 2010; Marshall et al., 2013). Albeit having smaller mean TSK score improvement in the P treatment group, this was not observed in the current RCT or two similar RCTs (Eadie et al., 2013; Hurley et al., 2015). Lumbar stabilization may mechanically decrease pain and reduce fear of movement. However, in teaching the exercise, the positive cognitive effects cannot be ignored when clinicians educate and explain the benefits of these exercises.

The fundamental use of exercise and advice/ pain education may have resulted in reductions in kinesiophobia. Exercise could have been either progressive walking exercise interventions and/or isometric lumbar stabilization. This was seen in this RCT and similar RCTs on cohorts with CLBP (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). In a RCT with 135 acute patients with sciatica, patients with a higher baseline level of kinesiophobia had particular benefit with reducing leg pain intensity at 12-month follow-up in response to physiotherapy (consisting of non-specific exercise and pain education) (Verwoed et al., 2015).

Mechanisms supporting improvements in kinesiophobia largely rely on increased movement, not necessarily a reduction in insufficiently active behaviour (Parker et al., 2017). The South African study showed that people with chronic pain tended towards insufficiently active behaviour (Parker et al., 2017). Therefore, consideration should be applied during treatment by monitoring the amount of increased PA advised to patients with CLBP. With increasing levels of PA, pain persistence behaviour may increase central sensitization and associated pain (Parker et al., 2017). Pain education, manual therapy and isometric lumbar stabilization exercises are seen in the current RCT and in RCTs in Lawford, Walters and Ferrar (2015). The difference in the current RCT from studies in previous systematic reviews on walking is that the usual care physiotherapy treatment group measured step count over 12 weeks. The usual care physiotherapy and exercise groups in RCTs in a previous systematic review did not record PA through step counts (Lawford, Walters and Ferrar; 2015). These intervention modalities may reduce kinesiophobia and not change insufficiently active status according to the <150-minute criteria, as was observed in the current RCT. This evidence concurs with that seen in the up-to-date systematic review showing a variety of conservative therapies are successful at reducing kinesiophobia in CLBP participants, regardless of change in insufficiently active behaviour according to the 150-minute criteria (Martinez-Calderon et al., 2020).

Several factors involved in exercise, PNE, and usual care physiotherapy could reduce kinesiophobia. There may be common relationships with kinesiophobia as in pain intensity and disability. As observed in the current RCT results, the PW treatment group presented with the greatest mean changes in TSK scores as well as pain and disability scores. Repeated visits may allow for additional assessment allowing for appropriate changes in therapy to improve outcomes. Increased number of taught exercises are seen to reduce pain and disability (Ferreira et al., 2010). This may support increased exposure to safe movement reducing fear avoidance. The results in the current trial would suggest this. The use of a pedometer and diary supporting graded and progressive movement exposure may be fundamental instruments in mitigating kinesiophobia. Patient education on CLBP management appears to be an important mechanism used to reduce fear of movement, common to many interventions studied previously (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). PNE has shown in combination with manual therapy and/ or exercise to decrease pain (Puentedura and Flynn, 2016). This may in turn support return to normal activity thereby reducing fear of movement. An example of PNE is use of 'the Back Book'. The Back Book was used in three RCTs using walking to treat CLBP (Eadie et al., 2013; McDonough et al., 2013; Hurley et al., 2015). Concurring with this evidence, a clinical trial on behavioural interventions using education and 'the Back Book' by participants with sub-acute LBP with graded exposure or graded activity when compared with treatment-based classification treatments showed reduction in fear avoidance beliefs in the graded exposure and treatment-based classification groups (George, et al., 2008). The treatment-based classification group received education on LBP and the graded exposure group utilized the Back Book. The graded activity group however also used the Back Book and did not show significant reductions in fear avoidance. This suggests that information on managing CLBP may amplify the effect on reducing kinesiophobia however not be limited to one source.

The number of physiotherapy visits was previously not analysed in systematic reviews comparing walking interventions on CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). In the current RCT the number of physiotherapy visits was a significant predictor of TSK score at 12-week follow-up. Participants attending one to two physiotherapy visits in the current RCT did not show statistically significant changes in TSK score at 12-week follow-up. In another study comparing physiotherapy to physiotherapy combined with a psychosocial intervention (PGAP), the number of physiotherapy visits ranged from two - four per week up to 10 weeks (Sullivan and Adams, 2010). Reductions of kinesiophobia occurred in both groups. Even though the number of physiotherapy visits were not analysed, both groups attended more than three physiotherapy visits. A recent systematic review of 61 RCTs analysed the effectiveness of conservative and surgical interventions in reducing fear, including kinesiophobia in people with CLBP (Martinez-Calderon et al., 2020). Mean time spent with participants was recorded in individual RCTs however the number of visits was never analysed as an independent variable. The

therapeutic relationship necessary to reduce kinesiophobia appears to require several visits to improve the outcome. Factors discussed previously such as exercise and/ or education on CLBP maybe fundamental requirements in the increased number of visits required for reduced kinesiophobia.

Mitigating fear associated with CLBP may vary depending on the mode in which the therapy is delivered. Therapy can be delivered by internet, telephone, or face to face. An up-to-date systematic review of fear of movement in individuals with CLBP did include mostly face to face therapy (Martinez-Calderon et al., 2020). However, two studies in this systematic review used internet based cognitive based therapy, both showing clinically significant reductions in fear avoidance beliefs over time (Carpenter et al., 2012; Chiauzzi et al., 2010). A RCT compared two internet-based treatments, an internet mediated walking program with support to internet log-ins for daily steps taken without support (Krein et al., 2013). The RCT found no difference between and within groups in fear avoidance at any time point from baseline to 12 months (Krein et al., 2013). These results differed to interventions using telephonic or face to face interactions. Therapeutic interactions via telephonic support showed reduced fear avoidance over time in walking interventions (Eadie et al., 2013; Hurley et al., 2015). Face to face interactions used for the exercise and usual care physiotherapy interventions also showed reduced fear avoidance over time. There were no statistically significant differences between groups in fear of movement (Eadie et al., 2013; Hurley et al., 2015). All treatment groups in the current RCT showed reductions in kinesiophobia, with all three treatment groups receiving face to face interactions. Due to different results shown between trials using walking for the outcome of fear avoidance behaviour, it may be worth considering the mode of optimal delivery when choosing a treatment.

Kinesiophobia may be worth clinical consideration when choosing an intervention for CLBP. It appears that there are a variety of treatment delivery options physiotherapists can choose from clinically, having varied value in the effect on TSK score. More than two physiotherapy visits appear to be beneficial for reducing kinesiophobia. Face to face or telephone supervision may be an avenue worth exploring with further research, as one RCT did not reduce fear of movement using internet mediated care (Krein et al., 2013).

Pain Catastrophizing

Patients who experience reductions in catastrophizing have demonstrated the potential for also minimising disability and pain outcome scores (Sullivan et al., 2006a; Adams et al., 2007). The pain catastrophizing cut - off score demonstrates pain catastrophizing behaviour with scores greater than 30/52 (Sullivan, Bishop and Pivik, 1995). A PCS score of 30 corresponds to the 75th percentile of the distribution of PCS scores in clinic samples of chronic pain patients (Sullivan, Bishop and Pivik,

1995). According to a cross sectional study of related MCIDs in 161 CLBP patients, a mean change in PCS score of 6.71 indicates a clinically significant threshold (Suzuki et al., 2020). In the current RCT, all three groups at baseline exhibited low pain catastrophizing scores (18/52). In a trial using walking as a treatment for CLBP and measuring fear avoidance, the authors mention a possible selection bias with participants being below the cut-off score for fear avoidance suggesting participants only entered the study who had a low fear of activity (Shnayderman and Katz-Leurer, 2013). The same may have occurred in the current RCT, where pain catastrophizers chose not to participate in this study.

There was no statistically significant difference in PCS scores between baseline and 12-week follow-up between treatment groups. All three treatment groups (P, PW, and W), however, showed statistically significant within group reduction in PCS scores from baseline to 12-week follow-up. The P and W treatment groups demonstrated similar mean reductions in PCS score from baseline to 12-week follow-up. The PW treatment group however, demonstrated the largest mean change in mean PCS score (-7.98). The confidence intervals indicate a plausible clinically significant reduction in mean PCS scores over time. (-10.9 to -5.07). NeuPSIG guidelines recommend including measurements of PCS score in chronic pain cohorts (Sullivan et al., 2006a; Haanpää et al., 2011). However, this RCTs' entire cohort at baseline presented with low levels of catastrophizing. There may be an over reliance on psychosocial factors, and some factors may not be the primary driver for the disorder (O'Sullivan, 2005). Future physiotherapy treatments may be informed that the results of this RCT were subject to a cohort with low PCS scores. Pain catastrophizing did not appear to be a psychosocial driver for CLBP in this cohort.

No studies in the reviews using walking interventions to treat CLBP measured pain catastrophizing (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). Unlike studies in these three reviews comparing walking interventions to usual care, the PGAP intervention used for CLBP however does use progressive walking programs and education to modify the behavioural variable of catastrophic thinking (Sullivan and Adams, 2010). This is concurred that pain and disability can be psychologically driven by mal-adaptive coping strategies such as pain catastrophizing (Nachemson, 1999). The results of the current RCT suggest further clinical research into the effects of a partly supervised walking intervention combined with physiotherapy if pain catastrophizing is suspected as a driving mechanism for the CLBP.

More than two physiotherapy visits had a significant effect on reducing PCS scores in the current RCT. Several factors may have influenced this variable. Repeated assessments may be an important mechanism in providing additional information to the therapists modifying additional treatment to improve the PCS outcome. Since pain catastrophizing is a maladaptive psychosocial variable, the

support and empathy during visits was not quantified but cannot be ignored in terms of the effects seen in placebo literature (Tavel, 2014). All three groups in this study were taught exercises which could be done independently and instructed not to aggravate pain intensity. As recognized in the PGAP intervention, increased exposure to movement has a positive effect on reducing pain catastrophizing (Sullivan and Adams, 2010). It is recognized that PNE can reduce pain and disability (Puentedura and Flynn, 2016). A trial on treating LBP using behavioural interventions and education, together with using 'the Back Book' by two treatment groups and education on LBP in the third group, showed similar reduction pain catastrophizing in all groups (George et. al, 2008). It is possible that pain education combined with treatments used in this RCT assisted the reduction in PCS scores.

Pain catastrophizing in this RCT is a modifiable variable. Studies included in reviews using walking and physiotherapy to treat CLBP did not measure pain catastrophizing (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). This novel evidence shows clinical potential.

5.4 Walking Intervention

The three previous systematic reviews comparing walking interventions to physiotherapy and exercise interventions have not compared manual physiotherapy with isometric lumbar stabilization or a pedometer driven walking program added to manual physiotherapy with isometric lumbar stabilization to a pedometer driven walking program (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). Despite there being no statistically significant difference in pain intensity, disability, kinesiophobia and pain catastrophizing between treatment groups, weekly step count differences were observed between the three treatment groups. However, the largest reduction in mean pain intensity scores were observed in the pedometer driven walking program combined with usual care physiotherapy group.

In three previous systematic reviews comparing walking interventions to physiotherapy and exercise interventions for CLBP, objectively measured PA was captured using pedometers, heart rate monitors and accelerometers in walking interventions (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). The current RCT demonstrated average weekly step counts in all three interventions whereby all participants used pedometers, regardless if a walking program was advised. This allowed for comparison of PA as a number of weekly steps taken between and within treatment groups. This was important in examining PA behaviour in CLBP participants which were exposed to three treatments. Previously, pedometers were used to measure the number of steps taken in the walking interventions, but not in the education or exercise or physiotherapy interventions (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Heart rate was used

in one RCT to grade PA intensity in both walking interventions (Karadeniz et al., 2014). Accelerometers were used for one week at baseline and follow-up to examine changes in PA (McDonough et al., 2013). Only one study used pedometers in the walking programme intervention and the control arm (Krein et al., 2013). It was novel to objectively compare weekly step counts between the current three treatment groups over time, while no statistically significant difference was seen in outcome measures between groups. Individual or multiple components of the three treatments may be responsible for the treatment effect.

Observing step counts in all three treatment groups indicated the decreased level of PA in chronic pain sufferers, specifically CLBP. This finding was corroborated in a recent South African study showing South African chronic pain populations exhibited low levels of PA but not specifying CLBP (Parker et al., 2017). Contextually, studies show lower levels of PA in CLBP populations (Björck-van Dijken et al., 2008; Vancampfort, Stubbs and Koyangi, 2017). However, it is inconclusive in studies of LBP (Heneweer et al., 2011). A study of 34129 adults ≥50 years, in six low-middle income countries, showed significant associations between CLBP and insufficiently active behaviour (Vancampfort, Stubbs and Koyangi, 2017). A Swedish study of 5798 adults suggested CLBP was associated with lower PA during leisure time (Björck-van Dijken et al., 2008). In contrast, a study of 5058 people demonstrated insufficient PA (<2.5 hours/week) was not associated with LBP (Hussain et al., 2016). Furthermore, and a systematic review between 1999-2009 noted inconsistent results for LBP with leisure time physical activities, sports, and physical exercise (Heneweer et al., 2011). It is possible that acute and sub-acute LBP may be precursors to insufficiently active behaviour associated with CLBP.

Ten thousand steps per day have become a benchmark for generic benefit to health outcomes (Tudor Locke and Bassett, 2004; ACSM, 2011; ACSM, 2013). The American College of Sports Medicine (ACSM) recommends a daily goal of 10,000 steps (ACSM, 2011; ACSM, 2013). Exercise is Medicine LBP guidelines recommend between 7000-9000 steps per day (Exercise is Medicine, 2019). It may be necessary to re-examine recommendations for people with CLBP during treatment. Participants in this trial increased their steps in the W and PW treatment groups. The P treatment group never increased their weekly step count over time. In the methodology of the current trial, steps were calculated as an average of steps taken per week. However, when observing the steps per week, the treatment groups randomised to the pedometer driven walking program never reached 10, 000 steps per day. The walking program advised was time based and not step based in the current RCT. Despite this, neither treatment group using the pedometer driven walking program achieved the weekly time goal set at the beginning of the RCT. No statistically significant difference between groups outcome measures was seen. However, when mean scores of outcome measures were compared over time, salient differences were noted. The greatest mean score changes were seen in

the PW treatment group, with the primary outcome of pain intensity demonstrating a plausible MCID in this treatment group. Increasing step counts above an unknown threshold may cause pain in this CLBP cohort. Pain neurophysiology studies expand on the process of sensitization which may have been responsible for increased pain with excess PA (Gangadharan and Kuner, 2013; Courtney, Fernández-de-Las-Peñas and Bond et al., 2017). Historical data suggests insufficiently active individuals or people living with chronic disease are shown to take between 3500-5500 steps/day, making the 10000 steps/day goals unachievable, risking failure and attrition (Iwane et al., 2000; Tudor-Locke and Myers, 2001b; Tudor-Locke et al., 2002a). This RCT together with outcomes from a study that examined feasibility to use pedometer driven walking for CLBP, suggests that step counts can be increased, associated with improved pain and disability outcomes (McDonough et al., 2013). However further investigation into optimal daily or weekly step counts, and strategies to increase them are required for CLBP treatment.

In the current RCT, weekly steps (from week 1-12) were divided by seven to show average daily step counts. Daily average step counts would amount to 3993 steps (P), 5565 steps (W) and 6258 steps (PW) over the trial period. Accelerometer data in a RCT in Ireland demonstrated baseline steps less than 8000 per day (McDonough et al., 2013). Average steps recorded in a South African Health study, which aimed to examine the relationship between average daily steps and health (determined by body composition), reflect similar numbers, where 312 adult participants took on average 6571(±3541) pedometer steps/day (Pillay et al., 2015). Another South African study aimed to compare PA levels of people with chronic pain (n=12) to a control group matched for age, gender and residential area (n=12) (Parker et al., 2017). Although the chronic pain participants were not exclusively those with CLBP, mean average daily step counts for people with chronic pain (2985) were lower than healthy controls (6409) (Parker et al., 2017). Both studies concurred with the current RCT where South Africans typically take less than 10,000 steps per day and exhibit low levels of PA (Pillay et al., 2015; Parker et al., 2017). Chronic pain therefore is associated with low PA levels observed in step counts in the current RCT and Parker et al., (2017). According to categorical data that classifies activity levels with outdated nomenclature, in the current RCT, after 12-weeks the P group was categorized as sedentary and W and PW groups as ‘low-active’ (Tudor Locke and Bassett, 2004). Despite objective differences, all three treatment groups remained insufficiently active taking less than 10,000 steps per day. This may indicate the walking intervention implementation was able to change activity levels in this cohort, but not able to reach 10,000 steps per day or more than 150 minutes of walking exercise per week. Since there was no follow-up after the 12-week intervention in the current RCT, it is unknown whether changed PA levels were sustained.

Furthermore, differences in PA were observed depending on pain phenotype in the entire cohort and not within treatment groups. Participants with neuropathic pain phenotype demonstrated lower step

counts compared to those with nociceptive pain phenotype over the trial (p=0.02). Albeit that both pain phenotypes in the entire cohort and not within treatment groups were insufficiently active, an activity level classification through steps taken categorized the nociceptive pain phenotype participants having greater activity levels (Tudor Locke and Bassett, 2004). Studies on CLBP have not yet measured pain phenotype differences in steps taken. However, in a study of 100 type-2 diabetic patients with diabetic neuropathy, having neuropathy was strongly associated with a lower step count (van Slotten et al., 2011). When using walking to treat CLBP, this association may assist clinicians in understanding the delayed progress of step counts in patients with CLBP phenotyped with neuropathic pain.

Methodologies in walking programs differ widely in studies included in systematic reviews using walking to treat CLBP (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). Advising walking programs in the current RCT and two others were based on the ACSM guidelines (Eadie et al., 2013, Hurley et al., 2015). Specific evidence-based application of walking exercise may be critical for advised walking programs used in CLBP since the current RCT and two others demonstrate not finding a statistically significant difference to comparator arms (Eadie et al., 2013; Hurley et al., 2015). However, the reporting of mean pain intensity score differences may indicate minimal clinical differences as observed in the current RCT. One RCT comparing a walking program and education to education only, utilized a graded pedometer-driven walking program around the 5A's model of health behaviour advice (McDonough et al., 2013). This advice was used previously for smoking cessation (Whitlock et al., 2002). The RCT was not fully powered, and despite both treatment groups showing improved pain and disability scores, the group with the walking program showed greater improvement. Thus far there is a lack of evidence in methods used whether one walking program is superior to another to use for patients with CLBP. The results in systematic reviews so far show walking to have no statistically significant difference when compared to other physiotherapy treatments and exercise programs (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). In the current RCT, the same walking program was used in two interventions, one with physiotherapy added to it, and there was no statistically significant difference in outcome measures between the treatment groups. However, a MCID was indicated for pain intensity in the PW group. This may suggest a walking program that is added to usual care physiotherapy has clinical potential for treatment of pain intensity in CLBP, but further exploration is required. The pedometer demonstrated that the walking program was not strictly adhered to by the PW treatment group. However, this combination of increased walking monitored by a pedometer combined with physiotherapy showed clinically meaningful results. An ideal walking program that is superior to others by means of outcome measures that are significantly reduced when compared to other treatments, has yet to be developed.

No trial used in the systematic reviews has compared the same walking program against itself and added an additional evidence-based treatment to it, such as physiotherapy while measuring step count in both (Hendrick et al., 2010; Lawford, Walters and Ferrar, 2015; Sitthipornvorakul et al., 2018). The pedometer-based walking intervention advised in the current RCT, for the W and PW groups was identical and increased in weekly increments based on ACSM guidelines. Both groups increased their step count over the 12-week intervention period indicating behavioural change in PA when exposed to a walking intervention. However, the PW group achieved higher weekly step counts compared to the W group. Weekly step count in both walking interventions did not increase to the extent proposed in the RCT methodology. Despite no statistically significant differences between treatment groups in pain intensity, disability, kinesiophobia and pain catastrophizing, the combination with usual care physiotherapy may have increased the supervisory capacity of the physiotherapists resulting in increased step counts. If secondary co-morbidities required increased step count, for example diabetes, cardiac conditions, or obesity, differences between groups step counts may require attention. The differences in step count/pacing between these two treatment groups may require further study to understand what factors motivated the increased step counts in the PW group despite being advised the same walking intervention. The W and PW groups increased their step number in week 1-12 however step number did not increase incrementally over the 12-weeks as was advised. Step count increased from baseline but increased minimally from week two onwards despite an upward trend. Several factors may have limited participant adherence to the incremental exercise dose increase. One factor may have been pain sensitization. Participants' pain may have increased, and the study methods were to instruct a reduction in walking until pain returned to ambient levels or lower. Furthermore, the PW group demonstrated a MCID in pain intensity at 12-week follow-up. The relationship of a MCID in pain intensity and greatest weekly step count may require further exploration. Reasons for not walking exactly as set out by the program were not recorded. Therefore pacing, effects of supervision, and goal setting may require further study in order to examine what effects varied amounts of walking have on CLBP. Since there is no gold standard of frequency, duration and intensity of walking which will be beneficial for CLBP outcomes, variances in pacing may require additional RCTs to investigate if these contributing factors are of greater benefit for outcome measures and treatment groups in this RCT. Evidence for exact amounts of exercise benefitting CLBP outcomes remains elusive. A review of 16 studies showed for health outcomes such as adiposity, blood lipids and psychological wellness, there is a paucity of evidence showing whether several bouts of exercise is as effective as one continuous bout (Murphy et al., 2009). A concurring view in cardiovascular benefits admits despite progress, understanding of how exercise dose across the spectrum affects cardiovascular health is needed (Wasfy and Baggish 2016). The same exists for CLBP. The current RCT proposed a methodology of a 10% weekly increase in time spent walking. This contrasts with steps counts set in participant consultation with physiotherapists in a RCT comparing a walking program with education on CLBP to education alone

(McDonough et al., 2013). It was noted in a few participants, that even with collaboration with their physiotherapists, step counts did not increase (McDonough et al., 2013). This behaviour was observed in the current RCT. The resistance to increase step counts in the current RCT requires further investigation as in McDonough et al., (2013). Perhaps setting of an activity goal is essential but how it is set is not important (McDonough et al., 2013). This was the case in the current RCT and three others setting goals for walking (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). It has been recommended that physiotherapists receive additional training in recognizing resistance to behavioural change and find strategies to better assist behaviour change (McDonough et al., 2013). Goal setting in clinical medicine is complex, involving several factors. However, unlike the current RCT, previous studies using walking for CLBP involved patient perspective on the amount of walking which should be completed (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Furthermore, methodological differences in how goals were set in the current RCT and walking interventions vary (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). The current RCT allowed for less than the advised duration to be done with no minimum of walking set to avoid increases in peripheral and central sensitization, although exercise goals (minutes per day) were pre-set. The findings of improved outcomes in this RCT concur with the American College of Sports Medicine position stand on prescribing exercise (Garber et al., 2011). Short, supervised bouts of exercise can produce significant health benefits (Garber et al., 2011). Unlike the current RCT, consultations regarding increases in walking were planned in three previous RCTs (McDonough et al., 2013; Eadie et al., 2013; Hurley et al., 2015). Regardless of these differences, these three RCTs and the current trial achieved improved pain and disability scores in the walking interventions. A RCT conducted in Norway used a self-exercise walking intervention which had no explicit incremental goals (Torstensen et al., 1998). Unlike exercise and physiotherapy comparator arms the self-exercise walking group showed no significant improvement in pain and disability outcomes. This strengthens the view that further research may be necessary for implementing optimal goals for CLBP walking interventions.

Individuals with CLBP may have varying levels of baseline PA, which may affect outcomes if exposed to walking or physiotherapy. To control for this variance individuals classified as insufficiently active (<150 minutes of exercise per week) were recruited for the current RCT. Some studies have also recruited participants classified as insufficiently active however using other methods to determine this. For example, one study used objective measures such as an ActivPAL ensuring less than 8500 steps per day at baseline (McDonough et al., 2013). Others have used a questionnaire to ensure “low” to “moderate” levels of PA measured using the International Physical Activity Questionnaire (IPAQ) (Hurley et al., 2015). Another study excluded participants doing PA more strenuous than slow paced walking more than twice a week (Shnayderman and Katz-Leurer, 2013). All of these studies attempted to include insufficiently active individuals; however varied

identifiers of baseline PA and varied objective measures of PA during the RCTs were used (McDonough et al., 2013; Shnayderman and Katz-Leurer, 2013; Hurley et al., 2015). Using similar treatment groups to the current RCT, in other walking RCTs (Eadie et al., 2013; Torstensen et al., 1998; Hurley et al., 2015), only Hurley et al., (2015) specified insufficiently active behaviour in the inclusion criteria. One fully powered RCT, however, did not recruit only insufficiently active individuals and used a self-paced walking intervention without objective measurement (Torstensen et al., 1998). Their results showed no significant improvements in pain or disability in the walking intervention. This may suggest supervised and graded dose walking as an exercise prescription for CLBP pain and disability outcomes may only be beneficial if the population is insufficiently active, but this requires further exploration. CLBP participants with stratified levels of insufficiently active behaviour may not respond equally to walking and physiotherapy interventions. Having a universal objective measure for study inclusion as well as objective measures throughout an intervention for insufficiently active and physically active CLBP populations may be worthwhile for matching treatment to prior levels of PA.

The current RCT used the definition of <150 minutes of exercise per week to recruit an insufficiently active population. This definition was used in a RCT comparing a pedometer driven walking intervention to usual care, and a paper outlining strategies to increase PA (Krein et al., 2013; Tuso, 2015). The methodological design for the current RCT was to increase PA to exceed the 150 minutes per week classification cut-off. When compared to other RCTs using pedometer-based walking interventions, the methodological design to supersede the threshold of 150 minutes was not used (Krein et al., 2013; McDonough et al., 2013). The results of the current RCT show that at no point in this 12-week RCT, did the mean value of the weekly time spent doing the walking intervention in the W or PW groups exceed 150 minutes per week. While PW and W treatment groups increased time spent walking, both remained insufficiently active according to this definition. It is not known if the results achieved in the W and PW groups would have remained the same had one or both groups increased step counts weekly in a linear manner and exceeded the 150 minute per week of advised walking threshold. This contrasted with the mean walking volume seen in the walking intervention of one RCT reporting 151.8 (±80.1) minutes per week (Hurley et al., 2015). A recent review on interventions to change PA classifies 150 minutes of PA per week or more as active, and 30-149 minutes as fairly active (National Institute for Health Research, 2019). The recognition of less than 150 minutes of PA per week may be necessary to encourage achievable exercise or PA goals for CLBP populations. This is particularly salient as the PW group demonstrated a MCID in pain intensity at the 12-week follow-up. Despite this, varied step counts between groups were seen over 12 weeks, and in context that all three treatment groups, and were insufficiently active at inclusion and remained insufficiently active at follow-up according to the 150-minute cut-off. Further research

may highlight varied outcomes depending on participant's levels of prior PA and appropriately advised PA or exercise.

Despite increased weekly step counts in W and PW groups in this RCT, all three treatment groups demonstrated no statistically between group differences. These results echo those seen in two RCTs, where walking interventions were not superior to manual therapy or even exercise therapy, not achieving statistically significant between-group differences in pain, disability, and fear avoidance behaviour (Hurley et al., 2015; Eadie et al., 2013). Former RCTs did not examine the number of physiotherapy visits as an independent variable, which perhaps requires further exploration. Torstensen et al., (1998) however showed manual therapy, and exercise therapy, to be superior to a self-exercise walking program. There was an absence of objective measurement of goal orientated walking, and CLBP education in Torstensen et al., (1998). This may furthermore explain the reduced effect in improving pain and disability in their self-exercise walking intervention as this was present in the current RCT. Evidence promotes exercise over manual therapy (Hayden et al., 2005b; Hendrick et al., 2010). However, it is seen that increasing activity with graded participation improves outcomes (Sullivan et al., 2006a; Ogunlana et al., 2018). The perspective in a review highlights the importance of pain education added to either manual therapy or exercise on outcome measures (Puentedura and Flynn, 2016). Despite no statistical between groups results in pain intensity, the results in this RCT suggest that education added to manual therapy as well as two types of exercise notwithstanding an isometric lumbar stabilization exercise as well as a pedometer driven walking exercise program demonstrate a MCID in pain intensity. Despite a trend of moving away from manual therapy towards the use of exercise, it does appear the presence of manual therapy with PNE is of clinical value (Puentedura and Flynn, 2016). Care, advice and education by means of supervision, face to face contact, telephone contact, or internet log-ins may have a positive effect on CLBP pain and disability outcomes despite walking program methodology (Torstensen et al., 1998; McDonough et al., 2013; Eadie et al., 2013; Krein et al., 2013; Shnayderman and Katz, 2013; Hurley et al., 2015).

5.5 Consideration of scientific and clinical implications

The current trial is novel and has several scientific and clinical implications.

Scientific recommendations

A scientific implication that has not been explored in previous studies using walking as a CLBP treatment but was significant in this RCT was number of physiotherapy visits. With the absence of NICE guidelines for the management of CLBP in South Africa, the methodology used in this RCT was implemented to carry out a level of evidence-based care associated with the number of visits. At

the time of trial design, the standard number of visits in NICE guidelines was between 9-10 physiotherapy visits. Due to the multiple factors involved in physiotherapy visits, this may be worth exploring at a practice, patient, or therapist level. Factors at a practice level including empathetic care, advice, and education by means of supervision, face to face contact, telephone contact, or internet logins may have a positive effect on CLBP pain and disability outcomes despite walking program or physiotherapy methodology. With the number of visits showing statistical significance in this RCT, it may be worth examining the relationship with number of visits and patient level characteristics further including patient gender, age, level of education, employment status and BMI associated to number of visits. Factors at therapist level associated with physiotherapy visits worth exploring may include additional LBP training, gender, age, and hours spent working per week. Additional examination into these variables may be of benefit with the knowledge that the number of visits has been associated with physiotherapist level variables. In typical South African physiotherapy private practice, patients can receive as many visits as they can afford in their own capacity. This includes the amount individual's medical insurance may cover. This was not the focus of this RCT but may warrant further attention. Future analysis of the effect of number of physiotherapy visits in context of private and public health care systems may be necessary.

According to the advised walking used in the current trial, increased walking used to treat CLBP independently, or in addition to usual care physiotherapy may require further exploration. The three treatment interventions used to improve the CLBP outcomes showed no statistically significant difference between groups. If walking exercise is to be studied as a treatment, with the hypothesis that it will be more effective than other CLBP treatments, perhaps a different method of walking intervention is to be used. Exploring a new methodology of walking intervention may require improvements on goal setting, and enhanced supervision understanding what factors affect pain intensity when walking. Although when the same walking program was added to usual care physiotherapy a MCID in pain intensity was observed at 12-week follow-up. Trialling this treatment against other forms of treatment for CLBP may prove to demonstrate statistically significant changes. In the current RCT, participants were not requested to record reasons why they were not able to maintain incremental time to walk in the W and PW treatment groups. If this data had been captured and analysed in the current RCT, potential obstacles to using the walking interventions, improved goal setting and improved pacing may have been demonstrated. Due to the chronic pain nature of the participants, pain sensitization may have been the main limiting factor in adhering to the walking programs. Solutions to this obstacle and others could be targeted. Future research into factors affecting participant adherence to walking interventions may prove useful in designing walking programmes for patients with CLBP which may show different results to the same outcomes.

Deciding on which measurement or outcome to use has scientific implications. Previous reviews using walking and physiotherapy to treat CLBP did not make use of some measures that were highlighted in this trial. Measuring pain catastrophizing, and expectation of pain intensity taken at baseline, may provide useful information in future studies of CLBP using the current treatments. Measuring pain catastrophizing may be worthy of exploration in future studies whose cohorts are typified by pain catastrophizing behaviour as it was a modifiable variable in the current RCT. Clinician management of patient expectations is an avenue requiring further exploration. Studies up until now have measured satisfaction with treatment. Literature on expectation of pain which affects outcomes infers a relationship (Koyama et al, 2005; Keltner et al 2013). A strength in this trial is that expectation should be studied before treatment of CLBP, since participants who expected lower pain intensity at baseline were likely to have lower pain intensity at the 12-week follow-up. Previous studies using walking to treat CLBP did not record participant expectation of pain. Using the same study design would be of interest examining pain outcomes between groups of high, moderate, or low levels of expectation of reduced pain intensity. Assessment of coping strategies with difficult demands was used in previous RCTs using walking and physiotherapy to treat CLBP. A measure of patient behaviour that was not used in this RCT was differences in coping strategies involved in pacing and goal setting. Self-efficacy improved in the walking and education as well as the education group, and participants in the walking and education group had 70% adherence to their weekly step targets (McDonough et al., 2013). Other authors postulated that participants underestimate the difficulty of changing their exercise behaviour at baseline (Krein et al., 2013; Hurley et al., 2015). Both authors reported reduced self-efficacy post RCT and suggested future investigations (Krein et al., 2013; Hurley et al., 2015). Outcome measures worth considering are Self efficacy scale, Chronic Pain Coping Inventory, and Patterns of Activity Measure-Pain scales (Jensen et al., 1995; Huijnen et al., 2011). The current RCT did not use these measures to analyse participants coping strategies with either self-efficacy or persisting/avoiding further activity. In the current RCT, patients in the PW treatment group were more likely to remain in the RCT compared to W and P treatment groups. Using the above measurements may help with understanding these differences in future studies.

Using the painDETECT to phenotype pain proved illuminating in observing distinct differences in pain phenotypes in this cohort. Potentially, future RCTs could use the painDETECT to phenotype pain to examine if other specific physiotherapy treatments are more efficient in treating CLBP pain phenotypes.

Clinical recommendations

There are several clinical implications to this novel research. Firstly, regardless of treatment intervention specifically applied in this RCT, the number of physiotherapy visits was statistically significant in changing the outcome measures used at follow-up. As expressed by a South African

study explaining that a poor understanding exists in treating CLBP, this trial should encourage physiotherapists to try a minimum of three treatments to see a positive change in outcomes. If the three treatment protocols are used clinically, patients and clinicians may expect to see positive changes following treatment exceeding three physiotherapy visits.

The only treatment group that demonstrated a MCID in pain intensity was the PW treatment group. Given the choice, advising patients to follow a pedometer driven walking program whilst attending usual care physiotherapy has clinical application when treating CLBP pain intensity.

No statistically significant difference between treatments was observed. Education about PNE together with walking or isometric lumbar stabilization appear to be common factors noticed in treatments associated with improved outcomes. These exercises and PNE may be encouraged in CLBP treatment.

Measuring what the patient expects their change in pain intensity to be prior to treatment, may be warranted. Physiotherapists may gain confidence with the association of positive expectation associated with reduced pain intensity post treatment. Understanding this may assist physiotherapy management treating pain intensity depending on the level of patient expectation.

Managing pain intensity remains a clinical priority. Although further study on coping mechanisms for managing pain intensity may be warranted in this cohort, caution may be necessary in selection of treatment and patient to prevent them discontinuing treatment. Patients with CLBP are more likely to continue with treatment using a pedometer-based walking program and usual care physiotherapy than a pedometer-based walking program alone. Physiotherapists using a NRS to measure pain intensity should be aware that patients with high levels of pain intensity may discontinue treatment at any point due to elevated pain levels. It may have been useful to use an outcome measure such as the Chronic Pain Coping Inventory or Patterns of Activity Measure-Pain understanding participants coping strategies with either persisting or avoiding further activity (Jensen et al., 1995; Huijnen et al., 2011).

Furthermore, using pain phenotyping at baseline may help clinicians understand varied patient progress during CLBP treatment. Physiotherapists and patients should not become despondent with smaller improvements seen in neuropathic pain phenotypes. Recognizing this process will help guide and manage patient management.

Chapter 6: Conclusion

Conservative treatments for CLBP are gaining traction since previously used treatments usual care physiotherapy and walking exercise. The two have not yet been combined and trialled against its constituent parts in a study. The primary objective of this RCT was to assess changes in pain intensity between baseline and 12-week follow-up between and within the following three treatment groups: usual care physiotherapy (P), a partly supervised pedometer-based walking intervention (W), and a combination of both (PW) in patients with nociceptive or neuropathic CLBP. Secondary objectives assessed changes in disability, kinesiophobia and pain catastrophizing between baseline and 12-week follow-up between and within the three groups. Uniquely, this RCT included pain phenotyping in the modelling processes. This RCT demonstrated that no statistically significant difference between groups was observed with respect to pain intensity, disability, kinesiophobia and pain catastrophizing. Respecting all four outcome measures, every treatment group showed improved within group scores at 12-week follow-up. The only treatment group showing a minimally clinically important difference in pain intensity at follow-up was the PW treatment group. Compared to the nociceptive pain phenotype, the neuropathic pain phenotype did predict a greater score in pain intensity and disability scores at 12-week follow-up.

Aside from the placebo effect or regression to the mean, common factors exist in the three treatment groups. Key points of this research highlight common factors between the three intervention groups and novel information regarding treatment of CLBP. With no statistically significant difference between the treatment groups at 12-week follow-up in outcome measures, similarities between groups were examined and four common factors between the treatment groups were identified in this RCT:

- the number face to face physiotherapy visits.
- The use of advice and education on CLBP.
- objective pedometer measurement of step count.
- implementation of an exercise intervention.

These common factors may have independently or in combination contributed to the results showing no statistically significant difference between treatment groups. All three interventions were represented by the presence of a physiotherapist in all treatment groups who gave advice and pain education on CLBP. All three interventions used a pedometer, possibly acting as a motivational factor, and recorded their steps and distance daily promoted for patient feedback. Lastly, all three interventions were treated with exercise (isometric lumbar stabilization exercise, a pedometer-based walking intervention, or both).

Novel information in the treatment of CLBP from the current RCT proposes the effect of the number of physiotherapy visits on pain intensity, kinesiophobia and pain catastrophizing outcomes. Acknowledging that expectation of pain intensity following these interventions at 12 weeks had not been studied, expecting a lower pain intensity at baseline was associated with a lower pain intensity at 12-week follow-up. This influence of expectation may have influenced all three treatment groups reduction in pain intensity. Additionally, neuropathic pain phenotype predicts greater pain and disability scores at follow-up.

The number of visits were shown to be a predictor of pain intensity, kinesiophobia and pain catastrophizing at 12-week follow-up. Up to now the number of physiotherapy visits has not been studied as an independent variable using the treatment groups in this RCT. There appears to be a threshold to how many treatments are necessary to obtain a significant improvement in outcome measures. The current trial showed three or more physiotherapy visits were statistically significant in improving pain intensity, kinesiophobia and pain catastrophizing scores at 12-week follow-up. Further exploration may provide insight into constituent factors in physiotherapy visits. Measurable factors can involve physiotherapist, patient, and practice levels. A more in-depth examination of these categories may facilitate insight into efficiency associated to number of physiotherapy visits for CLBP. The effect of physiotherapy visits may plateau and suggest the ideal number of visits necessary for improved outcomes. The physiotherapy visits were all done face to face in this RCT unlike the combination of face to face, telephone, or the internet in comparative RCTs. Depending on resources available, the number of physiotherapy visits may differ depending on the mode of interaction between physiotherapist and patient.

Associated with the number of visits was the teaching of an exercise to every treatment group. The novelty in this RCT was that all three treatment groups measured their steps throughout the week. When comparing weekly step count, only the usual care physiotherapy treatment group maintained consistent steps whilst the two treatment groups using the same walking program showed increased step counts. Despite statistically significant differences between treatment group step counts, there were no statistically significant differences in outcome measures between the three treatment groups at 12-week follow-up. The addition of the walking program used in this RCT, its absence, or used alone demonstrated no statistically significant difference in outcomes. The MCID in pain intensity shown in the PW treatment group is salient. In this RCT, step number can be compared between treatment groups. This suggests two courses of thought. One whereby results show statistical significance and the other denoting the clinical relevance of treatment. Firstly, the increased number of steps is not a requirement for a statistically significant difference in outcomes in this RCT, but only teaching of an exercise. The usual care physiotherapy treatment group demonstrated this

possibility by only performing isometric lumbar stabilization exercises. However, the addition of a pedometer driven walking program to usual care physiotherapy has clinical applications in the reduction of pain intensity. Additionally, the clinical implication of the MCID in pain intensity shown after 12-weeks in the PW group suggests clinical relevance to the use of this treatment. When choosing a walking intervention to reduce pain and disability from CLBP, the current RCT concurs with past evidence suggesting not all walking programs are equivalent in reducing CLBP. Up to now it appeared that designing a supervised or partly supervised progressive graded walking program whilst using a pedometer for objective measurement may demonstrate greater pain intensity score improvements compared to an unsupervised walking program with no graded dosage of walking and no objective measurement of PA. Objective measurement, and a form of structured program appear to be important factors although exercise pacing was not analysed in the current RCT walking programs. Possible alterations in frequency, duration and intensity of walking may be associated with different results in outcomes used. The motivation to improve health outcomes has been noticed when using pedometers previously. The ambient use of pedometers throughout the trial, in all three treatment groups, cannot be excluded as having a motivational effect on improving outcomes.

Another factor which was common to all three treatment groups was advice and education on CLBP. Although it was not standardized and based on manuscripts as seen in the Back Book, it may have contributed to all three treatment groups improved outcomes at 12-week follow-up. The effect of number of physiotherapy visits, inclusive of exercise, advice, and education on CLBP may include other factors that were not measured. The care effect or factors related to placebo have been studied previously, but never compared between the three interventions used in this trial. This RCT, as with others using walking and physiotherapy, may demonstrate that the care shown for CLBP patients has yet to be measured explicitly.

Unlike patient satisfaction, patient expectation was analysed in the entire cohort. The cohort demonstrated that those who expected lower pain intensity showed lower pain intensity scores. The data indicates that clinicians should acknowledge the patient's expectation of a treatment prior to administrating it. Treatments associated with patient's positive expectation of a reduction in pain intensity should be encouraged. Analysis in the current RCT was not performed on expectation of treatment group so no comment can be made on specific treatment. The cohort was from three medical settings. It is therefore likely their expectation for a result in reduced pain would be likely as participants were actively seeking treatment which may affect expectation. Acknowledging the patient's perspective on proposed treatment appears an important factor in future treatment.

The current RCT calls for future studies to expand on novel points. Due to no statistically significant between group differences seen, a comparison to other interventions would be reasonable. Two

interventions worthy of comparison are either evidence-based pain neuroscience education only, or usual care physiotherapy including massage and manipulation and excluding exercise and pain neuroscience education. If future trials are considered, advice and education should be standardized and based on evidence such as that found in the Back Book to improve reliability. Removing the isometric lumbar stabilization exercise and CLBP advice and education from the usual care physiotherapy treatment group in a future trial comparing to treatment groups including these factors, may suggest their importance as contributing factors to CLBP treatment differences. Due to the largest mean pain intensity score improvement seen in the PW treatment group, this treatment could be investigated further and trialled against other treatments not used in this trial.

Future studies should be constructed on examination of number of physiotherapy visits as an independent variable to treat outcomes of CLBP. Measuring how many visits are required before a treatment plateau is reached, including effect size of number of treatments is warranted for treatment groups used in this RCT. Further study can be done on patient, physiotherapist and practice level factors associated with number of treatments. Demographics factors influencing number of visits for example; age and gender could be influential factors in patient and physiotherapist characteristics. Physiotherapist experience may dictate the number of physiotherapist visits required to achieve a significant difference in outcome change. The same trial could be done in a public versus a private medical system to see if the number of physiotherapy visits required for a significant change in outcomes is the same between two system contexts. Furthermore, the mode in which physiotherapy visits would be worth further study due to differences seen in study methodologies. The number of physiotherapy visits could be compared using the same pedometer-based walking program to treat pain intensity, kinesiophobia and pain catastrophizing using face to face, telephone and internet logins as three different treatment groups. Treatment effect on outcomes and cost may differ depending on mode of interaction suggesting varied clinical application. Factors involved in patient care during physiotherapy visits that were not recorded in this RCT may be worth further examination such as empathy or warmth.

Improved measures between groups may prove useful for efficacy of a walking intervention. Pedometer data for 24 hours, including periods of rest may describe frequency, duration, and intensity of PA between treatment groups better. Study of these variables associated with daily pain intensity may give further insight into how these factors may affect pain sensitization. A future walking intervention with examination of participant differences in pacing recorded by frequency, duration and intensity may demonstrate a more efficient walking program for the treatment of CLBP. Recording differences in pacing associated with pain sensitization would be worthwhile to overcome obstacles due to pain preventing walking implementation. Following this, possibly using a mixed

methods methodology may provide valuable additional qualitative data exploring the application of the walking programs used in this trial.

South African physiotherapists have reported a lack of knowledge in understanding neurophysiological underpinnings of CLBP. Highlighting pain phenotyping, this trial is relevant for clinicians, patients and funders since neuropathic pain predicts greater pain intensity and disability scores at 12-week follow-up in this CLBP cohort. The relevance will allow patients to be tested using the painDETECT who receive CLBP treatment to understand that pain and disability outcomes may not be homogenous at baseline and following treatment between patients depending on pain phenotype. Acknowledging this allows for understanding of varied clinical outcomes between patients. Future trials may use the painDETECT together with more robust cut offs for neuropathic pain phenotype. Instead of using the 13-38 in this trial, using 19-38 would suggest a pain phenotype with an increased likelihood of neuropathic pain phenotype. An additional specialized clinical examination would add to making the clinical diagnosis of specific pain phenotypes. Future trials can test which treatment intervention is more efficient in reducing pain intensity, disability, kinesiophobia and pain catastrophizing in CLBP after phenotyping pain at baseline. This may prepare patients, physiotherapist's, and funders for differences in pain phenotypes noticed in this RCT. This trial only introduced the effect of pain phenotypes in the modelling process but was not powered to detect changes between groups based on phenotypes.

Recording what patients expect in pain intensity reduction from interventions in future trials is worthwhile. This trial showed that expecting a lower pain intensity was associated with achieving a reduced pain intensity at 12-week follow up in the entire cohort. Further study comparing intervention expectations may demonstrate cohort differences in expectation if treatment is revealed prior to baseline. It is known that expectation may be learned, so recording previous treatment experiences which were negative, ambiguous, or beneficial may be associated with varied changes in pain intensity at follow-up. This may add to understanding how treatment expectation can add value to proposed interventions.

A trial with the same treatments with a one-year follow-up may be beneficial to re-examine between and within group outcome measures. Examining whether the groups who used the walking program continued to have step counts greater than the usual care physiotherapy treatment group may provide insight into PA modification.

In future, studying the effects of the number physiotherapy visits on associated outcome measures would be a priority from findings of this study. A second point of departure would be to examine if other strategies could be employed to increase PA using walking and examine if these increases

promise improved outcome measure results. The clinical relevance of combining a partly supervised pedometer driven walking program with usual care physiotherapy is now better understood. Expectation appears intimately involved with pain intensity outcomes and is worthy of ethical appraisal in the clinical setting when treating patients. Lastly, physiotherapists may now have better understanding into varied responses in patients with CLBP based on pain phenotyping using questionnaires such as the painDETECT.

This trial adds novel approaches to considering number of visits when treating CLBP. With previous attention to types of treatments, the current trial suggests a new focus on how many times physiotherapists treat patients may affect outcomes. The clinical relevance of a pedometer driven walking program added to usual care physiotherapy is noted when treating CLBP pain intensity. The current RCT acknowledges diagnosing CLBP with neurophysiological and psychosocial underpinnings. In addition, perhaps a deeper understanding of treatment content delivered in future may require attention.

References

Adams, H., Ellis, T., Stanish, W.D., Sullivan, M.J., 2007. Psychosocial factors related to return to work following rehabilitation of whiplash injuries. *J. Occup. Rehabil*., 17(2), pp.305-315.

Ainsworth, B.E., Haskell, W.L., Whitt, M.C., Irwin, M.L., Swartz, A.M., Strath, S.J., O'Brien, W.L., Bassett, D.R. Jr, Schmitz, K.H., Emplaincourt, P.O., Jacobs, D.R. Jr, Leon, A.S., 2000. Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc*., 32(9 Suppl):S498-504.

Airaksinen, O., Brox, J.I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., Mannion, A.F., Reis, S., Staal, J.B., Ursin, H., Zanoli, G., 2006. Chapter 4: European guidelines for the management of chronic nonspecific low back pain. *Eur. Spine J.*, 15(Supplement 2), pp. s192-300.

Allegri, M., Montella, S., Salici, F., Valente, A., Marchesini, M., Compagnone, C., Baciarello, M., Manferdini, M.E., Fanelli, G., 2016. Mechanisms of low back pain: a guide for diagnosis and therapy. *F1000Research*, 5(F1000 Faculty Rev -1530), pp.1-13.

Alrwaily, M., Timko, M., Schneider, M., Stevans, J., Bise, C., Hariharan, K., Delitto, A., 2016. Treatment-Based Classification System for Low Back Pain: Revision and Update, *Phys. Ther*., 96(7), pp.1057–1066. Available from: https://doi.org/10.2522/ptj.20150345 [Accessed 20.02.2017]

Althoff, T., Sosič, R., Hicks, J. L., King, A. C., Delp, S. L., & Leskovec, J., 2017. Large-scale physical activity data reveal worldwide activity inequality. *Nature*, *547*(7663), pp. 336–339.

American College of Sports Medicine Consumer Information Committee., 2011. *ACSM info on …Starting a Walking Program.* [Online] Indianapolis: American College of Sports Medicine. Available from: https://uhs.berkeley.edu/sites/default/files/wellness-starting-a-walking-program.pdf [Accessed 10.01.2014]

American College of Sports Medicine. 2013. General Principles of Exercise Prescription – Exercising with Lower Back Pain. In: Pescatello, L., Arena, A., Riebe, D., Thompson, P., eds. *ACSM's Guidelines for Exercise Testing and Prescription*. 9th ed. Philadelphia: Lippincott Williams and Wilkins, pp.166-177.

America's Health Rankings, 2018. *Public Health Impact: Physical Inactivity*. [Online] USA: United Health Foundation. Available from: https://www.americashealthrankings.org/explore/annual/measure/Sedentary/state/ALL [Accessed on 01.03.2019]

Amir, R., Michaelis, M., Devor, M., 1999. Membrane Potential oscillations in Dorsal Root Ganglion Neurons: Role in Normal Electrogenesis and Neuropathic pain. *J. Neurosci*., 19(9), pp.8589-8596.

Amir, R., Devor, M., 2000. Functional cross-excitation between afferent A- and C- neurons in the dorsal root ganglia. *Neurosci.*, 95(1), pp.189-195.

Apkarian, A.V., Sosa, Y., Sonty, S., Levy, R.M., Harden, N., Parrish, T.B., Gitelman, D.R., 2004. Chronic Back Pain Is Associated with Decreased Prefrontal and Thalamic Gray Matter Density. *J. Neurosci.*, 24(46), pp.10410-10415.

Arendt-Nielsen, L., Svenson, P., 2001. Referred muscle pain: basic and clinical findings. *Clin. J. Pain.*, 17(1), pp.11-19.

Artus, M., van der Windt, D.A., Jordan, K.P., Hay, E.M., 2010. Low back pain symptoms show a similar pattern of improvement following a wide range of primary care treatments: a systematic review of randomized clinical trials. [Online]. *Rheumatology.*, 49(12), pp.2346–2356. Available from: https://doi.org/10.1093/rheumatology/keq245 [Accessed on: 03.11.2018]

Atlas,L.Y., Wager, T.D., 2012. Review: How expectations shape pain [Online] *Neurosci. Letters*.**,** 520(2), pp.140-148. Available from: http//: doi: 10.1016/j.neulet.2012.03.039. [Accessed on: 25.02.2017]

Attall, N., Bouhassira, D., Baron, R., Dostrovsky, J., Dworkin, R.H., Finnerup, F., Gourlay, G., Haanpaa, M., Raja, S., Rice, A.S.C., Simpson, D., Treede, R.D., 2011. Assessing symptom profiles in neuropathic pain clinical trials: Can it improve outcome ? [Online] *Eur. J. Pain*., 15(5), pp.441-43. Available from: https://doi.org/10.1016/j.ejpain.2011.03.005 [Accessed 14.05.2015]

Attal, N., Cruccu, G., Baron, R., Haanpää, M., Hansson, P., Jensen, T.S., 2010. EFNS guidelines on the pharmacological treatment of neuropathic pain: 2010 revision. *Eur. J. Neurol.*, 17(9), pp.1113-1123.

Avery, S., O'Driscoll, M., 2004. Randomised Controlled Trials on the Efficacy of Spinal Manipulation Therapy in The Treatment of Low Back Pain. *Phys. Ther. Rev*., 9(3), pp.146-152.

Bagwath Persad, L.A., Kamerman, P.R., Wadley, A.L., 2017. Predictors of Cold and Pressure Pain Tolerance in Healthy South African Adults. *Pain Medicine.*, 18(11), pp.2126–2137. Available from: https://doi.org/10.1093/pm/pnw291 [Accessed 09.03.2018]

Baker, G., Gray, S.R., Wright, A., Fitzsimons, C., Nimmo, M., Lowry, R., Mutrie, N., Scottish Physical Activity Research Collaboration (SPARColl). 2008. *Int. J. Behav. Nutr. Phys. Act*., 5(5), pp.44-49.

Balague, F., Troussier, B., Salimen, J., 1999. Non-specific low back pain in children and adolescents: risk factors. *Eur. Spine J*., 8(6), pp.429-438.

Barke, A., Preis, M.A., Schmidt-Samoa, C., Baudewig, J., Kröner-Herwig, B., Dechent, P., 2016. Neural Correlates Differ in High and Low Fear-Avoidant Chronic Low Back Pain Patients When Imagining Back-Straining Movements. *J. Pain*.,17(8), pp.930-943.

Baron, R., Binder, A., Attal, N., Casale, R., Dickenson, A.H., Treede, R.D., 2016. Neuropathic low back pain in clinical practice. *Eur. J. Pain*., 20(6), pp. 861-873.

Bassett, D.R., Wyatt, H.R., Thompson, H., Peters, J.C., Hill, J.O., 2010. Pedometer-Measured Activity in Health Behaviours in U.S. Adults. *Med. Sci. Sports Exerc*., 42(10), pp.1819-1825.

Blank, R.H., Burau, V., 2010. *Comparative Health Policy*. 3ed. Hampshire: Palgrave McMillan.

Beck, J.S., 1995. *Cognitive Therapy: Basics and Beyond*. New York-US: Guilford Press.

Behm, D.G., Drinkwater, E.J., Wilardson, J.M., Cowley, P.M., 2010. The use of instability to train the core musculature. *Appl. Physiol. Nut. Metab.*, 35 (1), pp.91-108.

Benedetti, F., Mayberg, H.S., Wager, T.D., Stohler, C.S., Zubieta, J., 2005. Neurobiological Mechanisms of the Placebo Effect. *J. Neurosci*., 25(45), pp.10390-10402. Available from: https//: DOI: https://doi.org/10.1523/JNEUROSCI.3458-05.2005. [Accessed: 02.04.2017]

Bennett, M.I., Attal, N., Backonja, M.M., Baron, R., Bouhassira, D., Freynhagen, R., Scholz, J., Tolle, T.R., Wittchen, H.U., Jensen, T.S., 2007. Using screening tools to identify neuropathic pain. *Pain.*, 127(3), pp.199-203.

Benning, T.B., 2015. Limitations of the biopsychosocial model in psychiatry. *Adv Med Educ Pract*., 6. pp.347-52. Available from: https//: doi: 10.2147/AMEP.S82937. eCollection 2015. [Accessed 17.06.17]

Bhatnagar, S.C., 2002. *Neuroscience for the Study of Communicative Disorders*. 2nd ed. Philadelphia: Lippincot Williams and Wilkins.

Bidzan-Bluma, I., Lipowska, M., 2018. Physical activity and cognitive functioning of children: A systematic review. *Int. J. Environ. Res. Public Health*., 15(4), pp.800.

Björck-van Dijken, C., Fjellman-Wiklund, A., Hildingsson, C., 2008. Low back pain, lifestyle factors and physical activity: a population based-study. *J Rehabil Med*., 40(10). pp.864-869.

Bleich, S.N., Ozaltin, E., Murray, C.J.L, 2009. How does satisfaction with the health-care system relate to patient experience? *Bull World Health Organ*., 87(4), pp.271-8.

Bogduck, N., 1995. The anatomical basis for spinal pain syndromes. *J. Manip. Physiolog. Therapeutics*. 18(9), pp.603-605.

Bokarius, A.V., Bokarius, V., 2010. Evidence-Based Review of Manual Therapy Efficacy in Treatment of Chronic Musculoskeletal Pain. *Pain Pract.*, 10(5), pp.451-458.

Boonstra, A.M., Stewart, R.E., KoKe, A.J., Oosterwijk, R.F., Swaan, J.L., Shreurs, K,M., Schiphorst Preuper, H.R., 2016. Cut-Off points for mild, moderate, and severe pain on the Numeric Rating Scale for Pain in Patients with Chronic Musculoskeletal Pain: Variability and Influence of Sex and Catastrophizing. [Online]. *Front Psychol.*, Available from: doi:3389/fpsyg.2016.01466 [Accessed on: 02.06.2019]

Boos, N., Lander, P.H., 1996. Clinical efficacy of imaging modalities in the diagnosis of low-back pain disorders. *Eur. Spine J.*, 5(1), pp.2-22.

Booth F.W., Roberts C.K., Laye M.J., 2012. Lack of exercise is a major cause of chronic diseases. *Compr. Physiol*., 2(2), pp.1143-1211.

Borenstein, D.G., Calin, A., 2012. *Low Back Pain (Fast Facts)*. 2nd ed. Abingdon: Health Press.

Borenstein, D.G., O'Mara, J.W. Jr., Boden, S.D., Lauerman, W.C., Jacobson, A., Platenberg, C., Schellinger, D., Wiesel, S.W., 2001. The value of magnetic resonance imaging of the lumbar spine to predict back pain in asymptomatic subjects: a seven-year follow up study. *J. Bone Joint Surg. Am*., 83(9), pp.1306-1311.

Bouhassira, D., Lantéri-Minet, M., Attal, N., Laurent, B., Touboul, C., 2008. Prevalence of chronic pain with neuropathic characteristics in the general population. *Pain.*, 136(3), pp.380-387.

Bousema, E.J., Verbunt, J.A., Seelen, H.A.M., Vlaeyen, J.W.S., Andre Knottnerus, J., 2007. Disuse and physical deconditioning in the first year after the onset of back pain. *Pain.*, 130, pp. 279-286.

Brady, T. J., 2011. Measures of self-efficacy: Arthritis Self-Efficacy Scale (ASES), Arthritis Self-Efficacy Scale-8 Item (ASES-8), Children's Arthritis Self-Efficacy Scale (CASE), Chronic Disease Self-Efficacy Scale (CDSES), Parent's Arthritis Self-Efficacy Scale (PASE), and Rheumatoid Arthritis Self-Efficacy Scale (RASE). *Arthritis Care Res (Hoboken)*. 2011;63 Suppl 11: S473-S485. Available from: https://onlinelibrary.wiley.com/doi/full/10.1002/acr.20567 [Accessed on 03.01.2018]

Bravata, D.M., Smith-Spangler, C., Sundaram, V., Gienger, A.L., Lin, N., Lewis, R., Stave, C.D., Olkin, I., Sirard, J.R., 2007. Using Pedometers to Increase Physical Activity and Improve Health: A Systematic Review. *JAMA.* 298(19), pp.2296–2304.

Bridges, D., Thompson, S.W.N., Rice, A.S.C., 2001. Mechanisms of neuropathic pain. *Br. J. Anaesth.*, 87(1), pp.12-26.

British Heart Foundation., 2017a. *New report assesses impact of physical inactivity on UK heart health and economy* [Online] London: British Heart Foundation. Available from: https://www.bhf.org.uk/news-from-the-bhf/news-archive/2017/april/new-report-assesses-impact-of-physical-inactivity-on-uk-heart-health-and-economy [Accessed on 08.04.2017]

British Heart Foundation., 2017b. Physical Inactivity Report 2017. [Online] London: British Heart Foundation. Available from: https://www.bhf.org.uk/-/media/filesresearch/heart-statstics/physcial-inactivity-report---mymarathon-final.pdf [Accessed 03.03.2018]

Bombardier, C., Hayden, J., Beaton, D.E., 2001. Minimal clinically important difference. Low back pain: outcome measures. *J. Rheumatol*., 28(2), pp.431-438.

Brodal, A., 1981. *Neurological Anatomy in Relation to Clinical Medicine*. 2nd ed. New York: Oxford University Press.

Bronfort, G., Haas, M., Evans, R.L., Leininger, B., Triano, J., 2010. The effectiveness of manual therapies: the UK evidence report. *Chiropr. Osteopat*., 18(1), pp.3.

Brown, C.A., 2004. The beliefs of people with chronic pain in relation to 'important' treatment components. *Eur. J. Pain*., 8(4), pp. 325-333.

Brukner, P., 2012. *Brukner & Khan's Clinical Sports Medicine.* 4th ed. Australia: McGraw- Hill Australia Pty Ltd.

Bruno M., Cummins S., Gaudiano L., Stoos J., Blanpied P., 2006. Effectiveness of two Arthritis Foundation programs: Walk with Ease, and YOU Can Break the Pain Cycle. *Clin. Interv. Aging*.,1(3), pp.295–306.

Bushnell, M.C., Apkarian, A.V., 2006. Representation of pain in the brain. In: McMahon, S.B., Koltzenburg, M., eds. *Wall and Melzck's Textbook of Pain.* 5th ed. London: Elsevier, pp.107-124.

Butler, D.S., Matheson, J., 2000. *The Sensitive Nervous System.* Adelaide Australia: Noigroup publications.

Butler, D.S., Moseley, G.L., 2013. *Explain Pain.* Adelaide, Australia: Noigroup publications

Bystad, M., Bystad, C., Wynn, R., 2015. How can placebo effects best be applied in clinical practice? A narrative review. *Psychol. Res. Behav. Manag*., 8, pp.41–45.

Byström, M.G., Rasmussen-Barr, E., Grooten, W.J., 2013. Motor control exercises reduces pain and disability in chronic and recurrent low back pain: a meta-analysis., 38(6):E350-8. Available from: https//: doi: 10.1097/BRS.0b013e31828435fb. [Accessed: 06.05.2017]

Callaghan, M., 1994. Evaluation of a back rehabilitation group for chronic low back pain in an outpatient setting. *Physiother*., 80(10), pp.677-681.

Callin, S., Bennet, M.I., 2008. Assessment of neuropathic pain. *CEACCP*.,8(6), pp.210-213.

Campbell, P., Foster, N. E., Thomas, E., & Dunn, K. M., 2013. Prognostic indicators of low back pain in primary care: five-year prospective study. *The Journal of pain: official journal of the American Pain Society.*, *14*(8), pp.873–883. Available from: https://: doi:10.1016/j.jpain.2013.03.013. [Accessed on 05.04.2018]

Campbell, J.N., Meyer, R.A., 2006. Mechanisms of Neuropathic Pain. *Neuron*., 52(1), pp.77-92.

Carette, S., Marcoux, S., Truchon, R., Grondin, C., Gagnon, J., Allard, Y., Latulippe, M., 1991. A controlled trial of corticosteroid injections into facet joint for chronic low back pain. *N. Engl. J. Med*., 325(14), pp.1002-1007.

Carpenter, K.M., Stoner, S.A., Mundt, J.M., Stoelb, B., 2012. An online self-help CBT intervention for chronic lower back pain. *Clin J Pain.*, 28, pp.14-22.

Caspersen, C.J., Powell, K.E., Christenson, G.M., 1985. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. *Public Health Rep*., 100(2), pp.126-131.

Casserley-Feeney, S.N., Daly, L., Hurley, D.A., 2012. The Access Randomized Clinical Trial of Public versus Private Physiotherapy for Low Back Pain. *Spine.*, 37(2), pp.85-96.

Celletti, C., Castori, M., La Torre, G., Camerota, F., 2013. Evaluation of Kinesiophobia and Its Correlations with Pain and Fatigue in Joint Hypermobility Syndrome/Ehlers-Danlos Syndrome Hypermobility Type. [Online] *BioMed Res. Internat*., Article ID 580460. Available from: https://doi.org/10.1155/2013/580460. [Accessed 12.04.2016]

Champ, C.E., Francis, L., Klement, R. J., Dickerman, R., Smith, R. P., 2016. Fortifying the Treatment of Prostate Cancer with Physical Activity. [Online] *Prostate Cancer.*, Available from: https://doi:10.1155/2016/9462975 [Accessed on 03.03.2019]

Chang, C.H., Cella, D., Clarke, S., Heinemann, A.W., Von Roenn, J.H. and Harvey, R., 2003. Should symptoms be scaled for intensity, frequency, or both? *Palliat. Support. Care., 1(1), pp.51-60.*

Chapman, C.R., Tuckett, R.P., Song, C.W., 2008. Pain and Stress in Perspective: Reciprocal Neural, Endocrine and Immune Interactions. *J Pain.*, 9(2), pp.122-145.

Charette, S.L. and Ferrell, B.A., 2007. Rheumatic diseases in the elderly: assessing chronic pain. *Rheum. Dis. Clinic N. Am., 33(1)*, pp.109-122.

Cherkin, D.C., Sherman, K.J., Deyo, R.A., Shekelle, P.G., 2003. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. *Ann Intern Med.*, 138(11), pp.898-906.

Chetty, S., Bhigjee, A.I., Baalbergen, E., Kamerman, P., Ouma, J., Raath, R., Raff, M., Salduker S., 2012. Clinical practice guidelines for management of neuropathic pain. Expert panel recommendations for South Africa. *SA Med. J*., 102(5), pp.312-325.

Chiauzzi, E., Pujol, L.A., Wood, M., Bond, K., Black, R., Yiu, E., Zacharoff, K., 2010. painACTION-back pain: a self-management website for people with chronic back pain. *Pain Med*., 11(7), pp.1044-1058.

Chiradeinant, A., Maher, C.G., Latimer, J., Stepkovitch, N., 2003. Efficacy of "therapist-selected" versus "randomly-selected" mobilization techniques for the treatment of low back pain: a randomized controlled trial. *Aust. J. Physiother*., 49(4), pp.233-241.

Cho, Y.K., Kim, D.Y., Jung, S.Y., Seong, J.H., 2015. Synergistic effect of a rehabilitation program and treadmill exercise on pain and dysfunction in patients with chronic low back pain. *J. Phys. Ther. Sci.*, 27, pp.1187-1190.

Chon, S.C., Chang, K.Y., You, J.S., 2010. The effect of the abdominal draw-in manoeuvre in combination with ankle dorsiflexion in strengthening the transverse abdominal muscle in healthy young adults: a preliminary, randomized, controlled study. *Physiother.*, 96(2), pp.130-136.

Chou, R., Clark, E., Helfand, M., 2003. Comparative efficacy and safety of long-acting oral opioids for chronic non-cancer pain: a systematic review. *J. Pain Symptom Manage*., 26(5), pp.1026-1048.

Chou, R., Qaseem, A., Snow, V., Casey, D., Cross, J.T.Jr., Scekelle, P., Owens, D.K., 2007. Diagnosis and treatment of Low Back Pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. *Ann. Intern. Med*., 147(7), pp.478-491.

Chou. R., 2010. Low back pain (chronic). *BMJ Clin Evid.* :1116.
PubMed PMID: 21418678; PubMed Central PMCID: PMC3217809. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3217809/pdf/2010-1116.pdf. [Accessed: 07.05.2015]

Cilliers, L., Maart, S., 2013. Attitudes, knowledge and treatment of low back pain amongst nurses in the Eastern Cape, South Africa. *Afr. J. Primary Health Care Fam. Med.*, *5*(1), pp.535.

Clemes, S.A., Hamilton, S.L., Griffiths, P.L., 2011. Summer to winter variability in the step counts of normal weight and overweight adults living in the UK. *J Phys Act Health*., 8(1), pp.36-44.

Clenzos, N., Naidoo, N., Parker, R., 2013. Physiotherapists' knowledge of pain: A cross-sectional correlational study of members of the South African Sports and Orthopaedic Manipulative Special Interest Groups. *SA J. Sports Med.*, 25(4), pp.95-100.

Coghill, N., Cooper, A.R., 2008. The effect of a home-based walking program on risk factors for coronary heart disease in hypercholeerolaemic men: a randomized controlled trial. *Prev. Med*., 46 (6), pp.545-551.

Cohen, J.W., 1988. Statistical Power Analysis for the Behavioural Sciences. 2nd ed. Hillsdale, NJ: Lawrence Erlbaum Associates.

Colcombe, S., Erickson, K.I., Scalf, P.E., Kim, J.S., Prakash, R., McAuley, E., Elavsky, S., Marquez, D.X., Hu, L., Kramer, A.F., 2006. Aerobic exercise training increases brain volume in aging humans. *J. Gerontol. A. Biol. Sci. Med. Sci.*, 61(11), pp.1166-1170.

Colcombe, S., Kramer, A.F., 2003. Fitness effects on the cognitive function in older adults: a meta-analytic study. *Psychol Sci*. 14(2), pp.125-130.

Colditz, G.A., Cannuscio, C.C., Frazier, A.L., 1997. Physical activity and reduced risk of colon cancer: implications for prevention. *Cancer Causes Control*., 8(4), pp.649–667.

Colloca, L., Miller, F. G., 2011. The nocebo effect and its relevance for clinical practice. *Psychosom. Med.*, 73(7), pp.598–603.

Costigan, M., Scholz, J., Woolf, C.J., 2009. Neuropathic Pain: A Maladaptive Response of the Nervous System to Damage. *Ann. Rev. Neurosci*., 32(1), pp.1-32.

Corder K., Brage, S., Ekelund, U., 2007. Accelerometers and pedometers: methodology and clinical application. *Curr Opin Clin Nutr Metab Care*., 10(5), pp.597-603.

Coudeyre, E., Rannou, F., Tubach, F., Baron, G., Coriat, F., Brin, S., Revel, M., Poiraudeau, S., 2006. General Practitioners' fear-avoidance beliefs influence their management of patients with low back pain. *Pain.*, 124(3), pp. 330-337.

Courtney, C. A., Fernández-de-Las-Peñas, C., Bond, S., 2017. Mechanisms of chronic pain - key considerations for appropriate physical therapy management. *J. Man. Manip. Ther.*, *25*(3), pp.118–127.

Crandall, J.P., Knowler, W.C., Kahn, S.E., Marrero, D., Florez, J.C., Bray, G.A., Haffner, S.M., Hoskin, M., Nathan, D.M., 2008. The prevention of type 2 diabetes. *Nat. Clin. Pract. Endocrinol. Metab*., 4(7), pp.382–393.

Crombez, G., Eccleston, C., Baeyens, F., Eelen, P., 1998. When somatic information threatens, Catastrophic Thinking Enhances Attentional Inference. *Pain.,* 75(2-3), pp.187-198.

Crombez, G., Eccleston, C., Van Damme, S., Vlaeyen, J.W., Karoly, P., 2012. Fear-avoidance model of chronic pain: the next generation. *Clin. J. Pain.* 28(6), pp.475-483.

Cruccu, G., Anand, P., Attal, N., Garcia-Larrea, L., Haanpää, M., Jorum, E., Serra., J., Jesen, T.S., 2004. EFNS guidelines on neuropathic pain assessment. *Eur. J. Neurol*., 11(3), pp.153-162.

Dagenais, S., Caro, J., Haldeman, S., 2008. A systematic review of lower back pain cost of illness studies in the United States and internationally. *Spine J.*, 8(1), pp.8-20.

Dankaerts, W., O'Sullivan, P., Burnett, A., Straker, L., Davey, P., Gupta, R., 2009. Discriminating healthy controls and two clinical subgroups of Nonspecific Chronic Low Back Pain Patients using Trunk Muscle activation and Lumbosacral Kinematics of Postures and Movements. *Spine.,* 34(15), pp.1610-1618.

Dankaerts, W., O'Sullivan, P., Straker, L.M., Burnett, A.F., Skouen, J.S., 2006. The inter-examiner reliability of a classification method for non-specific chronic low back pain patients with motor control impairments. *Man. Ther*., 11(1), pp.28-39.

Davis, K., 2006. Recent advances and future prospects in pain neuroimaging of acute and chronic pain. *Future Neurol*.,1(2), pp.203-213.

Davis, K.D., Flor, H., Greely, H.T., Iannetti, G.D., Mackey, S., Ploner, M., Pustilnik, A., Tracey, I., Treede, R.D., Wager, T.D., 2017, Brain Imaging Tests for chronic pain: medical, legal and ethical issues and recommendations. *Nat. Rev. Neurol*., 13(10), pp.624-638.

De Amici, D., Klersy, C., Ramajoli, F., Brustia, L., Politi, P., 2000. Impact of the Hawthorne effect in a longitudinal clinical study: the case of anesthesia. Control Clin Trials., 21(2), pp.103-14. Available from: doi:10.1016/s0197-2456(99)00054-9. PMID: 10715508. [Accessed 20.01.2021]

Dean, C.M., Duncan, P.W., 2016. Preparing the next generation of physical therapists for transformative practice and population management: an example from Macquarie University. *Phys Ther*., 23, pp.272- 274.

Delitto, A., Erhard, R.E., Bowling, R.W., 1995. A treatment-based classification approach to low back syndrome: identifying and staging patients for conservative treatment. *Phys Ther*., 75(6), pp.470-489

Delitto, A., 2005. Research in Low Back Pain: Time to Stop Seeking the Elusive "Magic Bullet", *Phys. Ther.*, 85(3), pp.206–208.

Deutscher, D., Horn, S.D., Dickstein, R., Hart, D.L., Smout, R.J., Gutvirtz, M., Ariel, I., 2009. Associations between treatment processes, patient characteristics, and outcomes in outpatient physical therapy practice. *Arch Phys Med Rehabil*., 90(8), pp.1349-1363.

Deveza, R.C., Elkins, M., Saragiotto, B.T., 2017. PEDro systematic review update: exercise for coronary heart disease. *Br. J. Sports Med*., 51(9), pp.755-756.

Devor, M., 2006. Response of nerves to injury in relation to neuropathic pain. In: McMahon, S.B., Koltzenburg, M., eds. *Wall and Melzack's Textbook of Pain.* 5th ed. London: Elsevier, pp.905-927.

Deyo, R.A., Rainville, J., Kent, D.L., 1992. What can the history and physical examination tell us about low back pain? *JAMA*., 268(6), pp.760-765.

Deyo, R.A., Weinstein, J.N., 2001. Low Back Pain. *N. Engl. J. Med*., 344(5), pp.363-370.

Diaz, E., Morales, H., 2016. Spinal Cord Anatomy and Clinical Syndromes. *Semin. Ultrasound CT MRI.*, 37(5), pp.360-371.

Diers, M., Koeppe, C., Diesch, E., Stolle, A.M., Holzl, R., Schiltenwolf, M., van Ackern, K., Flor, H., 2007. Central processing of acute muscle pain in chronic low back pain patients: an EEG mapping study. *J. Clin. Neurophysiol*., 24(1), pp.76-83.

Di Iorio, A., Abate, M., Guralnik, J. M., Bandinelli, S., Cecchi, F., Cherubini, A., Corsonello, A., Foschini, N., Guglielmi, M., Lauretani, F., Volpato, S., Abate, G., Ferrucci, L., 2007. From chronic low back pain to disability, a multifactorial mediated pathway: the InCHIANTI study. [Online] *Spine*, 32(26), E809–E815. Available from: doi:10.1097/BRS.0b013e31815cd422. [Accessed: 12.07.2017]

Dionne, C.E., Dunn, K.M., Croft, P.R.,2006. Does back pain prevalence really decrease with increasing age? A systematic review. *Age and Ageing.*, Volume 35(3), pp.229–234.

Dionne, C.E., Korff, M., Koepsell, T., Deyo, R., Barlow, William., Checkoway, H., 2001. Formal education and back pain: A review. *Journal of epidemiology and community health.*, 55, pp.455-68.

D'Mello, R., Dickenson, R., 2008. Spinal cord mechanisms of pain. *Br. J. Anaesth*., 101(1), pp.8-16.

Dobson, J.L., McMillan, J., Li, L., 2014. Benefits of exercise intervention in reducing neuropathic pain. *Frontiers Cellular Neurosci.*, 8(102), pp.1-9.

Dombrowski, S.U., Sniehotta, F.F., Avenell, A., Johnston, M., Maclennan, G., Araújo-Soares, V., 2012. Identifying active ingredients in complex behavioural interventions for obese adults with obesity-related co-morbidities or additional risk factors for co-morbidities: a systematic review. *Health Psychol Rev*., 6(1), pp.7–32.

Donnachie, C., Wyke, S., Mutrie, N., Hunt, K., 2017. 'It's like a personal motivator that you carried around wi' you': Utilising self-determination theory to understand men's experiences of using pedometers to increase physical activity in a weight management programme. *The International Journal of Behavioral Nutrition and Physical Activity, 14*(1), pp.61.

Dray, A., Perkins, M., 1993. Bradykinin and inflammatory pain. *Trends Neurosci*., 16(3), pp.99-104.

Dreisinger, T. E., 2014. Exercise in the management of chronic back pain. *Ochsner J.*, *14*(1), pp.101–107.

Dubin, A.E., Patapoutian, A., 2010. Nociceptors: the sensors of the pain pathway. *J. Clin. Invest*., 120(11), pp.3760-3772.

Duffy, R.L., 2010. Low back pain: an approach to diagnosis and management. *Prim. Care*., 37(4), pp.729-741.

Dworkin, R.H., Turk, D.C., Wyrwich, K.W., Beaton, D., Cleeland, C.S., Farrar, J.T., Haythornthwaite, J.A., Jensen, M.P., Kerns, R.D., Ader, D.N. and Brandenburg, N., 2008. Interpreting the clinical importance of treatment outcomes in chronic pain clinical trials: IMMPACT recommendations. *J. Pain*, 9(2), pp.105-121.

Eadie, J., van de Water, A.T., Lonsdale, M.A., Tully, M.A., van Mechelen, W., Boreham, C.A., Daly, L., McDonough, S.M., Hurley, D.A., 2013. Physiotherapy for sleep disturbance in people with chronic low back pain: Results of a feasibility Randomized Controlled Trial. *Arch. Phys. Med. Rehabil*., 94(11), pp.2083-2092.

Eccleston, C., Crombez, G., 1999. Pain demand attention: a cognitive-affective model of the interruptive function of pain. *Psychol. Bull*., 125(3), pp.356-366.

Eccleston, C., Crombez, G., Aldrich, S., Stannard, C., 1997. Attention and somatic awareness in chronic pain. *Pain.*, 72(1-2), pp.209-215.

Edwards, R.R., Smith, M.T., Stonerock, G., Haythornthwaite, J.A., 2006. Pain-related catastrophising in healthy women is associated with greater temporal summation of a reduced habituation to thermal pain. *Clin. J. Pain*., 22(8), pp.730-737.

Edwards, R. R., Dworkin, R. H., Sullivan, M. D., Turk, D. C., & Wasan, A. D., 2016. The Role of Psychosocial Processes in the Development and Maintenance of Chronic Pain. *J. Pain.*, 17(9 Suppl), T70–T92. Available from: https://doi.org/10.1016/j.jpain.2016.01.001 [Accessed 11.08.2017]

Ekman, E.F., Koman, L.A., 2004. Acute pain following musculoskeletal injuries and orthopaedic surgery. *J. Bone Joint Surg*., 86(6), pp.1316-1327.

Ellingsen, D.M., 2014. *Central modulation of affective touch, pain, and emotion in humans*. Thesis (Doctural), Gothenberg Universitet, Sweden.

El Sissi, W., Arnaout, A., Chaarani, M.W., Fouad, M., El Assuity, W., Zalzala, M., Dershaby, Y.E., Youseif, E., 2010. Prevalence of neuropathic pain among patients with chronic-low back pain in the Arabian Gulf Region assessed using the Leeds assessment of neuropathic symptoms and signs pain scale. *J. Int. Med. Res*., 38(6), pp.2135-2145.

Elvey, R., O'Sullivan, P., 2004. *A contemporary approach to manual therapy: Modern Manual Therapy*. 3rd ed. Amsterdam: Elsevier

Engelke, K., Kemmler, W., Lauber, D., Beeskow, C., Pintag, R., Kalender, W.A., 2006. Exercise maintains bone density at spine and hip EFOPS: a 3-year longitudinal study in early postmenopausal women. *Osteoporos Int.*17(1), pp.133–142.

Ernst, E., 1999. Massage therapy for low back pain: a systematic review. *J. Pain Symptom Manage*., 17(1), pp.65-69.

Ernst, E., Fialka, V., 1994. Ice freezes pain? A review of the clinical effectiveness of analgesic cold therapy. *J. Pain Symptom Manage.,* 9(1), pp.56-59.

Etnier, J.L., Nowell, P.M., Landers, D.M., Sibley, B.A., 2006. A meta-regression to examine the relationship between aerobic fitness and cognitive performance. *Brain Res. Rev*., 52(1), pp.119–130.

Exercise is Medicine 2019. Being Active When You Have Low Back Pain, Exercise is Medicine. American College of Sports Medicine. Available from: https://www.exerciseismedicine.org/assets/page_documents/EIM_Rx%20for%20Health_Low%20Back%20Pain.pdf [Accessed 02.07.2019]

Fairbank, J.C., Pynsent, P.B., 2000. The Oswestry Disability Index. Spine., 25(22), pp.2940-2953.

Farber, K., Wieland, L.S., 2016. Massage for Low-back Pain. *Explore (NY)*.,12(3), pp.215-217.

Farrar,J.T., Portenoy, R.K., Berlin, J.A., Kinman, J.L., Strom, B.L., 2000. Defining the clinically important difference in pain outcome measures. *Pain*., 88, pp.287-294.

Farrar, J.T., Young, J.P., Jr, LaMoreaux, L., Werth, J.L., Poole, R.M., 2001. Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain*, 94, pp.149-158.

Fatoye, F., Gebrye, T., Odeyemi, I., 2019. Real-world incidence and prevalence of low back pain using routinely collected data. *Rheumatol Int*., 39(4), pp.619-626.

Faul, F., Erdfelder, E., Lang, A.G., Buchner, A., 2007. G* Power 3: A flexible statistical power analysis program for the social, behavioural, and biomedical sciences. *Behav Res. Meth.*, 39(2), pp.175-191.

Forero, D.E., Gómez, A., Comparison of measurement models based on expectations and perceived performance for the satisfaction study in health services. *Suma Psicológica*., 24(2), pp.87-96.

Ferrari, S., Villafane, J.H., Berjano, P., Vanti, C., Monticone, M., 2018. How many physical therapy sessions are required to reach a good outcome in symptomatic lumbar spondylolisthesis? A retrospective study. *J. Bodywork Move. Ther.*, 22(1), pp.18-23.

Ferreira, M.L., Smeets, R.J.E.M., Kamper, S.J., Ferreira, P.H., Machado, L.A.C., 2010. Can we explain heterogeneity among randomized clinical trials of exercise for chronic back pain? A Meta-regression analysis if randomized controlled trials. *Phys. Ther*., 90(10), pp.1383-1403.

Feizerfan, A., Sheh, G., 2014. Transition from acute to chronic pain. *CEACCP*., 15(2), pp.1-5.

Field, T., 2014. Massage therapy research review. *Complement Ther. Clin. Pract*., 20(4), pp.224-229.

Fielding R.A., Guralnik, J.M., King, A.C., Pahor, M., McDermott, M.M., Tudor-Locke, C., Manini, T.M., Glynn, N.W., Marsh, A.P., Axtell, R.S., Hsu, F.C., Rejeski, W.J. LIFE study group., 2017. Dose of physical activity, physical functioning, and disability risk in mobility-limited older adults: Results from the LIFE study randomized trial. *PloS one*, *12*(8), e0182155. Available from: https//: doi:10.1371/journal.pone.0182155 [Accessed 09.10.2018]

Fillingim, R.B.., Hastie, B.A., Ness, T.J., Glover, T.L., Campbell, C.M., Staud, R., 2005. Sex-related psychological predictors of baseline pain perception and analgesic responses to pentazocine. *Biol. Psychol.*, 65(1 SPEC. ISS.), pp.97-112.

Finnerup, N.B., Jensen, T.S., 2006. Mechanism of Disease: mechanism-based classification of neuropathic pain- a critical analysis. *Nat. Clin. Pract. Neurol.*, 2(2), pp.107-115.

Finnerup, N.B., Sindrup, S.H., Jensen, T.S., 2007a. Chronic neuropathic pain: mechanisms, drug targets and measurement. *Fundam. Clin. Pharmacol*., 21(2), pp.129-136.

Finnerup, N.B., Sorensen, L., Biering-Sorensen, F., Johannesen, I.L., Jensen, T.S., 2007b. Segmental hypersensitivity and spinothalamic function in spinal cord injury pain. *Exp. Neurol*., 207(1), pp.139-149.

Fishbain, D.A., Cole, B., Lewis, J.E., Gao, J., 2014. What is the Evidence that Neuropathic Pain is present in Chronic Low Back Pain and Soft Tissue Syndromes? An Evidence-Based Structure Review. *Pain Med*., 15(1), pp.4-15.

Fleming, K.C. and Volcheck, M.M., 2015. Central sensitization syndrome and the initial evaluation of a patient with fibromyalgia: a review. *Rambam Maimonides Med. J.*,6(2), pp.e0020. Available from: https//: doi:10.5041/RMMJ.10204 [Accessed 15.05.2016]

Flor, H., Braun, C., Elbert, T., Birbaumer, N., 1997. Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neurosci. Lett.*, 224(1), pp.5-8.

Follick, M.J., Ahern, D.K. and Laser-Wolston, N., 1984. Evaluation of a daily activity diary for chronic pain patients. *Pain*, 19(4), pp.373-382.

Ford, J.J., Hahne, A.J., 2013. Complexity in the physiotherapy management of low back disorders: Clinical and research implications. *Man. Ther*., 18(5), pp.438-442.

Förster, M., Mahn, F., Gockel, U., Brosz, M., Freynhagen, R., Tölle, T. R., & Baron, R., 2013. Axial low back pain: one painful area--many perceptions and mechanisms. [Online] *PloS one*, *8*(7), e68273. Available from: https//: doi: 10.1371/journal.pone.0068273 [Accessed 11.04.2015]

Fourie, M., 2019. Reaching across sectors. *Physio SA.*, 32(4), pp.7-8. Available from https://www.saphysio.co.za/media/178324/2018-no-04-physiosa-june.pdf [Accessed 21.09.2019]

Freburger, J.K., Carey, T.S., Holmes, G.M., Wallace, A.S., Castel, L.D., Darter, J.D., Jackman, A.M., 2009. Exercise prescription for chronic back or neck pain: who prescribes it? who gets it? What is prescribed? *Arthritis Rheum*., 61(2), pp.192-200.

Freynhagen, R., Baron, R., 2009. The evaluation of neuropathic components in low back pain. *Curr. Pain Headache Rep*., 13(3), pp.185-190.

Freynhagen, R., Baron, R., Gockel, U., Tölle, T.R., 2006. painDETECT: a new screening questionnaire to identify neuropathic components in patients with back pain. *Curr. Med. Res. Opin*., 22(10), pp.1911–1920.

Froud, R., Patterson, S., Eldridge, S., Seale, C., Pincus, T., Rajendran, D., Fossum, C., Underwood, M., 2014. A systematic review and meta-synthesis of the impact of low back pain on people's lives. [Online] *BMC Musculoskel. Disord*., 15 (50). Available from: https://www.doi: 10.1186/1471-2474-15-50. [Accessed 03.03.2015]

Fry, D.E., 2018. "The Hawthorne Effect Revisited." Diseases of the Colon & Rectum., 61 (1), pp. 6-7.

Furlan, A.D., Brosseau, L., Imamura, M., Irvin, E., 2002. Massage for low-back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. *Spine*., 27(17), pp.1896-1910.

Furlan, A.D., Imamura, M., Dryden, T., Irvin, E., 2008. Massage for low-back pain.[Online]. *Cochrane Database Syst Rev.*, 4, Art. No.:CD001929. Available from: https//: doi: 10.1002/14651858.CD001929.pub2. [Accessed 09.03.2015]

Gangadharan, V., Kuner, R., 2013. Pain hypersensitivity mechanisms at a glance. *Dis. Model. Mech.*, 6(4), pp.889-895.

Garber, C.E., Blissmer, B., Deschenes, M.R., Franklin, B.A., Lamonte, M.J., Lee, I.M., Nieman. D.C., Swain, D.P., American College of Sports Medicine., 2011. American College of Sports Medicine position stand. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Med Sci Sports Exerc*., 43(7), pp.1334-1359

Garcia-Larrea, L., Peyron, R., 2013. Pain matrices and neuropathic pain matrices: a review. *Pain* ®., 154(Supplement 1), pp.S29-S43.

Gatchel, R.J., Peng, Y.B., Peters, M.L., Fuchs, P.N., Turk, D.C., 2007. The biopsychosocial approach to chronic pain: scientific advances and future directions. *Psychol. Bull*., 133(4), pp.581-624.

Gazzi, M.L., Maher, C.G., Hancock, M.J., Kamper, S.J., McAuley, J.H., Stanton, T.R., 2014. Predicting response to motor control exercises and graded activity for patients with low back pain: preplanned secondary analysis of a randomized controlled trial. *Phys. Ther*., 94(11), pp.1543–1554

Gerriten, J.K.W., Vincent, A.J.P.E. 2016. Exercise improves quality of life in patients with cancer: a systematic review and meta-analysis of randomised controlled trials. *Br. Sports Med*., 50(13), pp.796-803.

George, S.Z., Valencia, C. and Beneciuk, J.M., 2010. A psychometric investigation of fear-avoidance model measures in patients with chronic low back pain. *J. Ortho. Sports Phys.Ther.*, 40(4), pp.197-205.

George, S.Z., Zeppieri Jr, G., Cere, A.L., Cere, M.R., Borut, M.S., Hodges, M.J., Reed, D.M., Valencia, C. and Robinson, M.E., 2008. A randomized trial of behavioural physical therapy interventions for acute and sub-acute low back pain (NCT00373867). *Pain.*, 140(1), pp.145-157.

Giesecke, T., Gracely, R.H., Grant, M.A., Nachemson, A., Petzke, F., Williams, D.A., Clauw, D.J., 2004. Evidence of Augmented Central Pain Processing in Idiopathic Chronic Low Back Pain. *Arthritis Rheum.*, 50(2), pp.613-623.

Gifford, L.S., Butler, D.S., 1997. The integration of pain sciences into clinical practice. *J. Hand Ther*., 10(2), pp.86-95.

Goh, S.L., Persson, M.S.M., Stocks, J., Hou, Y., Lin, J., Hall, M.C., Doherty, M., Zhang, W., 2019. Efficacy and potential determinants of exercise therapy in knee and hip osteoarthritis: A systematic review and meta-analysis. [Online], in press. *Ann. Phys. Rehabil. Med.*, Available from: https://doi: 10.1016/j.rehab.2019.04.006. [Accessed 22.05.2019]

Goldby, L.J., Moore, A.P., Doust, J., Trew, M.E., Marion, E., 2006. A randomized control trial investigating the efficacy of musculoskeletal physiotherapy on chronic low back pain disorders. *Spine*., 31(10), pp.1083-1093.

Goodin, B.R., McGuire, L., Allshouse, M., Stapleton, L., Haythornthwaite, J.A., Burns, N., Mayes, L.A., Edwards, R.R., 2009. Associations between catastrophising and endogenous pain-inhibitory processes: sex differences. *J Pain*., 10(2), pp.180-190.

Goosens, M., Vlaeyen, J., Hidding, A., Kole-Snijders, A., Evers, S., 2005. Treatment expectancy affects the outcome of cognitive-behavioural interventions in chronic pain. *Clin. J. Pain*., 21(1), pp.18-26.

Gordon, R., Bloxham, S., 2016. A systematic review of the effects of Exercise and Physical activity on Non-Specific Chronic Low Back Pain. *Healthcare.*, 4(2), pp.1-22.

Gosling, A.P., 2013. Physical therapy action mechanisms and effects on pain management. *Rev. Dor. São Paulo*., 13(1), pp.65-70.

Gracey, J.H., McDonough, S.M., Baxter, G.D., 2002. Physiotherapy management of low back pain: a survey of current practice in northern Ireland. *Spine (Phila Pa 1976).*, 27(4), pp.406-411

Gracely, R.H., Geisser, M.E., Giesecke, T., Grant, M.A.B., Petzke, F., Williams, D.A., 2004. Pain catastrophising and neural responses to pain among persons with fibromyalgia. *Brain.*, 127(4), pp.835-843.

Grachev, I.D., Fredrickson, B.E., Apkarian, A.V., 2000. Abnormal brain chemistry in chronic back pain: an in vivo proton magnetic resonance spectroscopy study. *Pain.*, 89(1), pp.7-18.

Greaves, C.J., Sheppard, K.E., Abraham, C., Hardeman, W., Roden, M., Evans, P.H., Schwarz, P., Yhe IMAGE study Group., 2011. Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions. *BMC Public Health* 11**,** 119. Available from: https://doi.org/10.1186/1471-2458-11-119 [Accessed on 20.12.2020]

Groenendijk, J.J., Swinkels, I.C.S., de Bakker, D., Dekker, J., van den Ende, C.H.M., 2007. Physical therapy of low back pain has changed. *Health Policy*., 80(3), pp.492-499.

Gustin, S.M., Peck, C.C., Wilcox, S.L., Nash, P.G., Murray, G.M., Henderson, L.A., 2011. Different Pain, Different Brain: Thalamic Anatomy in Neuropathic and Non-Neuropathic Chronic Pain Syndromes. *J. Neurosci*., 31(16), pp.5956-5964.

Guthold, R., Louazani, S.A., Riley, L.M., Cowman, M.J., Bovet, P., Damasceno, A., SAmbo, B.H., Tesfaye, F., Armstrong, T.P., 2011. Physical activity in 22 African countries: results from the World Health Organization STEPwise approach to chronic disease risk factor surveillance. *Am J Prev Med*., 41(1), pp.52-60.

Haanpää, M., Attal, N., Backonja, M., Baron, R., Bennett, M., Bouhassira, D., Cruccu, G., Hansson, P., Haythornthwaite, J.A., Iannetti, G.D., Jensen, T.S., Kauppila, T., Nurmikko, T.J., Rice A.S.C., Rowbotham, M., Serra, J., Sommer, C., Smith, B.H., Treede, R.D., 2011. NeuPSIG guidelines on neuropathic pain assessment. *Pain.*, 152(1), pp.14–27.

Haefeli, M. and Elfering, A., 2006. Pain assessment. *Eur. Spine J.*, 15(1), pp.S17-S24. Available from: https//: doi:10.1007/s00586-005-1044-x [Accessed 22.05.2016]

Hägg, O., Fritzell, P. and Nordwall, A., 2003. The clinical importance of changes in outcome scores after treatment for chronic low back pain. *Eur. Spine J.*, 12(1), pp.12-20.

Hall, T.M., Elvey, R.L., 2004. Management of mechanosensitivity of the nervous system in spinal pain syndromes. In: Boyling, J., Jull, G., eds. *Grieve's Modern Manual Therapy*. 3rd ed. Edinburgh: Churchill Livingstone, pp.413-31.

Hall, A.M., Ferreira, P.H., Maher, C.G., Latimer, J., Ferreira, M.L., 2013. The influence of the therapist-patient relationship on treatment outcome in physical rehabilitation: A systematic review. *Phys. Ther*., 90(8), pp.1099-1110.

Hancock, M.J., Maher, C.G., Latimer, J., McAuley, J.H., 2006. Selecting an appropriate placebo for a trial of spinal manipulative therapy. *Aus. J. Physiother*., 52(2), pp.135–138.

Harris, T., Kerry, S.M., Victor, C.R., Ekelund, U., Woodcock, A., IIiiFFe, S., Whincup, P.H., Beighton, C., Ussher, M., Limb, E.S., David, L., Brewin, D., Adams, F., Rogers, A., Cook, D.G., 2015. A primary care nurse-delivered walking intervention in older adults: PACE (Pedometer Acceleraometer Consultation Evaluation)- Lift Cluster Randomized Controlled Trial. [Online]. *PLOS Med*., 12(2), pp. e1001783. Available from: https://doi.org/10.1371/journal.pmed.1001783 [Accessed on 02.03.2015]

Hartvigsen, J., Morsø, L., Bendix, T., & Manniche, C., 2010. Supervised and non-supervised Nordic walking in the treatment of chronic low back pain: a single blind randomized clinical trial. *BMC musculoskeletal disorders*., *11*, 30. Available from: https://doi.org/10.1186/1471-2474-11-30 [Accessed on 10.19.2016]

Harvey, E., Burton, A.K., Moffett, J.K., Breen, A., UK BEAM trial team., 2003. Spinal manipulation for low-back pain: a treatment package agreed to by the UK chiropractic, osteopathy and physiotherapy professional associations. *Man Ther*., 8(1), pp.46-51.

Hassan, A.E., Saleh, H.A., Baroudy, Y.M., 2005. Prevalence of neuropathic pain among patients suffering from chronic lower back pain in Saudi Arabia. *Neurosci*., 25(10), pp.51-55.

Hatano, Y., 1993. Use of the pedometer for promoting daily walking exercise. *ICHPER.*, 29(1), pp.4-8.

Hayden, J.A., Maurits, D.C., van Tulder, W., 2005a. Systematic review: Strategies for using exercise therapy to improve outcomes in chronic low back pain. *Ann. Inter. Med.*, 142(9), pp. 776-785.

Hayden, J.A., van Tulder, M.W., Malmivaara A.V., Koes, B.W., 2005b. Meta-analysis; exercise therapy for nonspecific low back pain. *Ann. Intern. Med*., 142(9), pp.765-775.

Hayes, S.C.,Bisset, R.T., Korn, Z., Zettle, R.D., Rosenfarb, I.S., Cooper, L.D., Grundt, A.M., 1999. The impact of acceptance versus control rationales on pain tolerance. *Psychol. Record*., 49(1), pp.33-47.

Haythornthwaite, J.A., Clarke, M.R., Pappagallo, M., Raja, S.N., 2003a. Pain coping strategies play a role in the persistence of pain in post herpetic neuralgia. *Pain*., 106(3), pp.453-460.

Haythornthwaite, J.A., Wegener, S., 2003b. Factors associated with willingness to try different pain treatments for pain after spinal cord injury. *Clin. J. Pain*., 19(1), pp.31-38.

Helliwell, P.S., Bennet, R.M., Littlejohn, G., Muirden, Wigley, R.D., 2003. Towards epidemiological criteria for soft-tissue disorders of the arm. *Occ. Med*., 53(8), pp.313-319.

Henchoz, Y., Kai-Lik So, A., 2008. Exercise and nonspecific low back pain: a literature review. *Joint Bone Spine*., 75(5), pp.533-539.

Hendrick, P., Te Wake, A.M., Tikkisetty, A.S., Wulff, L., Yap, C., Milosavljevic, S., 2010. The effectiveness of walking as an intervention for low back pain: a systematic review. *Eur Spine J*., 19(10), pp.1613-1620.

Hendrick, P., Milosavljevic, S., Hale., L., Hurley, D.A., McDonough, S., Ryan, B., Baxter, G.D., 2011. The relationship between physical activity and low back pain outcomes: a systematic review of observational studies. *Eur Spine J*, 20, pp.464-474.

Heneweer, H., Staes, F., Aufdemkampe, G., van Rijn, M., Vanhees, L., 2011. Physical activity and low back pain: a systematic review of recent literature. *Eur. Spine J.*, 20(6), pp.826–845.

Heymans, M.W., de Vet, H.C., Bongers, P.M., Knol, D.L., Koes, B.W., van Mechelen, W., 2006. The effectiveness of high intensity versus low intensity back schools in an occupational setting: a pragmatic randomized controlled trial. *Spine.*, 31(10), pp.1075-1082.

Heyneman, N.E., Fremouw, W.J., Gano, D., Kirkland, F. and Heiden, L., 1990. Individual differences and the effectiveness of different coping strategies for pain. *Cog. Ther. Res.*, 14(1), pp.63-77.

Hicks, G.E., Fritz, J.M., Delitto, A., McGill, S.M., 2005. Preliminary development of a predication rule for determining which patients with low back pain will respond to a stabilization exercise program. *Arch. Phys. Med. Rehabil*., 86(9), pp.1753-1762.

Hildebrandt, V.H., 1987. A review of epidemiological research on risk factors of low back pain. In: Buckle P.E., editor. *Musculo-skeletal disorders at Work*. London: Taylor & Francis, pp.9–16.

Hillman, C.H., Erickson, K.I., Kramer, A.F., 2008. Be smart, exercise your heart: exercise effects on brain and cognition. *Nat. Rev. Neurosci*., 9(1), pp.58–65.

Hirvensalo, M., Telama, R., Schmidt, M.D., Tammelin, T.H., Xiaolin, Y., Magnussen, C.G., Vkari, J.S., Raitakari, O.T., 2011. Daily steps among Finnish adults: variation by age, sex and socioeconomic position. *Scand. J. Public Health*., 39(7), pp.669-677.

Hodges, P.W., Moseley, G.L., 2003. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. *J. Electromyogr. Kinesiol*., 13(4), pp.361-370.

Hoheisel, U., Reuter, R., de Freitas, M.F., Treede, R.D., Mense, S., 2013. Injection of nerve growth factor into low back muscle induces long-lasting latent hypersensitivity in rat dorsal horn neurons. *Pain.*, 154(10), pp.1953-1960.

Hoy, D., Brooks, P., Blyth, F., Buchbinder, R., 2010. The epidemiology of low back pain. *Best Pract. Res. Clin. Rheumatol*., 24(6), pp.769-781.

Hruschak, V., Cochran, G., 2018. Psychosocial predictors in the transition from acute to chronic pain: a systematic review. *Psychol Health Med.*, 23(10), pp.1151-1167.

Hsieh, C.Y., Phillips, R.B., Adams, A.H., Pope, M.H., 1992. Functional outcomes of low back pain: comparison of four treatment groups in a randomized controlled trial. *J Manipulative Physiol Ther.* 15(1), pp.4-9.

Humphreys, B.K., P. M. Irgens, P.M., 2002. The Effect of a Rehabilitation Exercise Program on Head Repositioning Accuracy and Reported Levels of Pain in Chronic Neck Pain Subjects. *J. Whiplash & Related Disord*., 1(1), pp.99-112.

Hurley, D.A., Tully, M.A., Lonsdale, C., Boreham, C.A.G., van Mechelen, W., Leslie, D., Aódan, T., McDonough, S.M., 2015. Supervised walking in comparison with fitness training for chronic back pain in physiotherapy: results of the SWIFT single-blinded randomized controlled trial (ISRCTN17592092). *Pain.*, 156(1), pp.131-147.

Huijnen, I.P.J., Verbunt, J.A., Roelofs, J., Goossens, M., Peters, M., 2009. The disabling role of fluctuations in physical activity in patients with chronic low back pain. *Eur J Pain.*, 13, pp.1076–1079.

Huijnen, I.P., Verbunt, J.A., Peters, M.L., Smeets, R.J., Kindermans, H.P., Roelofs, J., Goossens, M., Seelen, H.A., 2011. Differences in activity-related behaviour among patients with chronic low back pain. *Eur J Pain*., 15(7), pp.748-755.

Huijnen, I.P., Rusu, A.C., Scholich, S., Meloto, C.B., Diatchenko, L., 2015. Subgrouping of low back pain patients for targeting treatments: evidence from genetic, psychological, and activity-related behavioural approaches. *Clin. J. Pain*, 31(2), pp.123-132.

Hussain, S. M., Urquhart, D. M., Wang, Y., Dunstan, D., Shaw, J. E., Magliano, D. J., Wluka, A.E., Cicuttini, F. M., 2016. Associations between television viewing and physical activity and low back pain in community-based adults: A cohort study. *Medicine*, *95*(25), e3963. Available from: https//: doi:10.1097/MD.0000000000003963 [Accessed 11.04.2018]

Hush, J. M., Nicholas, M., & Dean, C. M., 2018. Embedding the IASP pain curriculum into a 3-year pre-licensure physical therapy program: redesigning pain education for future clinicians. *Pain reports*, *3*(2), e645. Available from: https://doi:10.1097/PR9.0000000000000645 [Accessed: 03.12.2018]

Ilnyckyj, A., Shanahan, F., Anton, P.A., Cheang, M., Bernstein, C.N., 1997. Quantification of the placebo response in ulcerative colitis. *Gastroenterol*., 112(6), pp.1854-1858.

Inani, S.B., Selkar, S.P., 2013. Effect of core stabilization exercises versus conventional exercises on pain and functional status in patients with non-specific low back pain: A randomized clinical trial. *J Back Musculoskelet. Rehabil.*, 26(1), pp.37-43.

Institute for Health Metrics and Evaluation (IHME). 2018. *Findings from the Global Burden of Disease Study 2017*.[Online] Seattle, WA: IHME Available from: http://www.healthdata.org/sites/default/files/files/policy_report/2019/GBD_2017_Booklet.pdf [Accesed 02.04.2019]

Iwane, M., Arita, M., Tomimoto, S., Satani, O., Matsumoto, M., Miyashita, K., Nishio, I., 2000. Walking 10,000 steps/day or more reduces blood pressure and sympathetic nerve activity in mild essential hypertension. *Hypertens. Res.*, 23(6), pp.573-580.

Iwata K., Kamo H., Ogawa A., Tsuboi Y., Noma N., Mitsuhashi Y., Tiara, M., Koshikawa, N., Kitagawa, J., 2005. Anterior cingulate cortical neuronal activity during perception of noxious thermal stimuli in monkeys. *J. Neurophysiol.*, 94, pp.1980–1991.

Jackson, M.A., Simpson, K.H., 2006. Chronic back pain. *CEACCP*., 6(4), pp.152-155.

Jackson, T., Wang, Y., Wang, Y., Fan, H., 2014. Self-efficacy and chronic pain outcomes: a meta-analytic review. *J Pain.*, 15(8), pp.800-814.

Jamison, R.N. and Edwards, R.R., 2012. Integrating pain management in clinical practice. *J. Clin. Psychol.med. Setting*, 19(1), pp.49-64.

Jay, G.W., Barkin, R.L., 2014. Neuropathic pain: Aetiology, pathophysiology, mechanisms and evaluations. *Dis. Mon*., 60(1), pp.6-47.

Jensen, M. P., Turner, J. A., Romano, J. M., and Strom, S. E., 1995. The chronic pain coping inventory: development and preliminary validation. *Pain.*, 60(2), pp. 203–216.

Jensen, M.P., Turner, J.A., Romano, J.M., 2001. Changes in Beliefs, Catastrophising and Coping are associated with Improvement in Multidisciplinary pain Treatment. *JCCP*., 69(4), pp.655-662.

Jette, A.M., Smith, K., Haley, S.M., Davis, K.D., 1994. Physical therapy episodes of care for patients with low back pain. *Phys. Ther*., 74(2), pp.101 - 115.

Julius, D., Basbaum, A.I., 2001. Molecular mechanisms of nociception. *Nature.*, 413(2001), pp.203-210.

Julius, D., McCleskey, E.W., 2006. Cellular and molecular properties of primary afferent neurons. In: McMahon, S.B., Koltzeburg, M., eds. *Wall and Melzack's Textbook of Pain.* 5th ed. London: Elsevier, pp.35-48.

Kääpä, E.H., Frantsi, K., Sarna, S., Malmivaara, A., 2006. Multidisciplinary group rehabilitation versus individual physiotherapy for chronic nonspecific low back pain: a randomized trial. *Spine*., 31(4), pp.371-376.

Kaki, A.M., El-Yaski, A.Z., Youseif, E., 2005. Identifying neuropathic pain among patients with chronic low-back pain: use of the Leeds Assessment of Neuropathic Symptoms and Signs pain scale. *Reg. Anesth. Pain Med*., 30(5), pp.422-428.

Kalso, E., Edwards, J.E., Moore, R.A., McQuay, H.J., 2004. Opioids in chronic non-cancer pain: systematic review of efficacy and safety. *Pain*., 112(3), pp.372-380.

Kamerman, P.R., Bradshaw, D., Laubscher, R., Pillay-van Wyk, V., Gray, G.E., Mitchell, D., & Chetty, S., 2020. Almost 1 in 5 South African adults have chronic pain: a prevalence study conducted in a large nationally representative sample. *Pain.*, *161*(7), pp.1629–1635. Available from: https://doi.org/10.1097/j.pain.0000000000001844 [Accessed: 10.08.2020]

Kang, M., Marshall, S.J., Barreira, T.V., Lee, J.O., 2009. Effect of pedometer-based physical activity interventions: a meta-analysis. *Res. Q. Exerc. Sport*. 80(3), pp.648-655.

Kaptchuk, T.J., Kelley, J.M., Conboy, L.A., Davis, R.B., Kerr, C.E., Jacobson, E.E., Kirsch, I., Schyner, R.N., Nam, B.H., Nguyen, L.T., Park, M., Rivers, A.L., McManus, C., Kokkotou, E., Drossman, D.A., Goldman, P., Lembo, A.J., 2008. Components of placebo effect: randomised controlled trial in patients with irritable bowel syndrome. *Brit. Med. J*., 336(7651) pp.999-1003.

Karadeniz, M., Dandinoglu, T., Yazicioglu, K., Tan, A.K., 2014. Assessment and comparing of Effectiveness of Overground and Treadmill walking on Chronic Low Back Patients [Online] *Int. J. Phys. Med. Rehabil*., 181., pp. 2-6. Available from: https://doi:10.4172/2329-9096.1000181 [Accessed 02.03.2014]

Karayannis, N. V., Jull, G. A., Hodges, P. W., 2012. Physiotherapy movement based classification approaches to low back pain: comparison of subgroups through review and developer/expert survey.[Online] *BMC Musculoskel. Disord., 13*, 24. Available from: https//: doi:10.1186/1471-2474-13-24 [Accessed 15.03.2015]

Katsura, Y., Ueda,S.Y., Yoshikawa,T., Usui,T., Orita,K., Sakamoto,H., Sotobayashi,D., Fujimoto,S., 2011. Effects of aquatic exercise training using new water-resistance equipment on trunk muscles, abdominal circumference, and activities of daily living in elderly women. [Online] *Int. J. Sport Health Sci*., Available from: https://www.jstage.jst.go.jp/article/ijhs/advpub/0/advpub_201112/_pdf [Accessed 02.03.2015]

Katz P., Margaretten M., Gregorich S., Trupin L., 2018. Physical activity to reduce fatigue in rheumatoid arthritis: a randomized controlled trial. *Arthritis Care Res*.,70(1), pp.1–10.

Keefe, F.J., Abernethy, A.P., Campbell, L.C., 2005. Psychological approaches to understanding and treating disease related pain. [Online] *Annu. Rev. Psychol*., 56, pp.601–630. Available from: https://doi10.1146/annurev.psych56091103.070302. [Accessed on 02.03.2015]

Keltner, J.R., Furst, A., Fan, C., Redfern, R., Inglis, B., Fields, H.L., 2006. Isolating the modulatory effect of expectation on pain transmission: a functional magnetic resonance imaging study. *J Neurosci*., 26(16), pp.4437-4443.

Kim, J.D., Oh, H.W., Lee, J.H., Cha, J.Y., Ko, I.G., Jee, Y.S., 2013. The effect on inversion traction on pain sensation, lumbar flexibility and trunk muscles strength in patients with chronic low back pain. *Isokinet. Exerc. Sci*., 21(3), pp.237-246.

Kimura, F., Shimizu, K., Akama, T., Akimoto, T., Kuno, S., Kono, I., 2006. The effects of walking exercise training on immune response in elderly subjects. *Int. J. Sport Health Sci*., 4(Special-Issue-2-2006), pp.508-514.

Koes, B.W., Assendelft, W.J., van der Heijden, G.J., Bouter, L.M., 1996. Spinal manipulation for low back pain. An updated systematic review of randomized clinical trials. *Spine*., 21(24), pp.2860-2871.

Koes, B.W., van Tulder, M., Lin, C.C.W., Macedo, L.G., McAuley, J., Maher, C., 2010. An updated overview of clinical guidelines for the management of non-specific low back pain in primary care. *Eur. Spine J*., 19(12), pp.2075-2094.

Koes, B.W., van Tulder, M., Ostelo, R., Burton, K., Waddell, G., 2001. Clinical guidelines for the management of Low Back Pain in Primary Care: An International Comparison. *Spine*., 26(12), pp.2504-2513.

Koes, B.W., van Tulder, M.W., Thomas, S., 2006. Diagnosis and treatment of low back pain. *BMJ*., 332(7555), pp.1430-1434.

Koldaş Doğan, S., Sonel Tur, B., Kurtaiş, Y., Atay, M.B., 2008. Comparison of three different approaches in the treatment of chronic low back pain. *Clin Rheumatol*., 27(7), pp.873-881.

Koltzenburg, M., 1999. The changing sensitivity in the life of the nociceptor. *Pain*., 82(Supplement)1, pp.S93-102. Available from: https//: doi:10.1016/s0304-3959(99)00142-6 [Accessed 11.06.2016]

Kori, S.H., Miller, R.P., Todd, D.D., 1990. Kinesiophobia: A new view of chronic pain behaviour. *Pain Manage*., 3, pp.35–43.

Kosek, E., Cohen, M., Baron, R., Gebhart, G.F., Mico, J.A., Rice, A.S., Rief, W. and Sluka, A.K., 2016. Do we need a third mechanistic descriptor for chronic pain states?. *Pain*, 157(7), pp.1382-1386.

Koyama, T., McHaffie, J. G., Laurienti, P. J., Coghill, R. C., 2005. The subjective experience of pain: where expectations become reality. *Proceedings of the National Academy of Sciences of the United States of America.*, 102(36), pp.12950–12955.

Kramer, A.F., Hahn, S., Cohen, N.J., Banich, M.T., McAuley, E., Harrison, C.R., Chason, J., Vakil, E., Bardell, L., Boileau, R.A., Colcombe, A., 1999. Ageing, fitness and neurocognitive function. *Nature*., 400(6743), pp.418–419.

Krause, S.J., Backonja, M.M., 2003. Development of a neuropathic pain questionnaire. *Clin J Pain*. 19(5), pp.306-14.

Kregel, J., Meeus, M., Malfliet, A., Dolphens, M., Danneels, L., Nijs, J., Cagnie, B., 2015. Structural and Functional brain abnormalities in chronic low back pain: A systematic review. *Semin Arthritis Rheum*., 45(2), pp.229-237.

Krein, S.L., Metreger, T., Kadri, R., Hughes, M., Kerr, E.A., Piette, J.D., Kim, H.M., Richardson, C.R., Hollerman, R., 2013. Pedometer-based Internet mediated intervention for Adults with Chronic Low Back Pain: randomized control trial, *J. Med. Internet Res*., 15(8), pp.e181. Available from: https://doi.org/10.2196/jmir.2605 [Accessed 02.04.2016]

Kruger, P.E., Billson, J.H., 2012. The effect of conservative versus an aggressive-progressive exercise programme on chronic low back pain and disability. *AJPHERD*., Supplement (March), pp.120-131.

Kujala, U.M., Sarna, S., Kaprio, J., Koskenvuo, M., 1996. Asthma and other pulmonary diseases in former elite athletes. *Thorax*., 51(3), pp.288–292.

Kucyi, A., Davis, K.D., 2015. The dynamic pain connectome. *Trends Neurosci*., 38(2), pp.86-95.

Kumanyika, S.K., Obarzanek, E., Stettler, N., Bell, R., Field, A.E., Fortmann, S.P., Franklin, B.A., Gillman, M.W., Lewis, C.E., Poston, W.C., Stevens, J., Hong, Y., 2008. Population-based prevention of obesity: the need for comprehensive promotion of healthful eating, physical activity, and energy balance: a scientific statement from American Heart Association Council on Epidemiology and Prevention, Interdisciplinary Committee for Prevention (formerly the expert panel on population and prevention science). *Circulation*., 118(4), pp.428–464.

Kumar, K., Abbas, M., Rizvi, S., 2012. The use of spinal cord stimulation in pain management. *Pain Manag*., 2(2), pp.125-134.

Kumar, S., Beaton, K., Hughes, T., 2013. The effectiveness of massage therapy for the treatment of nonspecific low back pain: a systematic review of systematic review. [Online] *Int. J. Gen. Med*. 6, pp.733-741. Available from: https://doi10.2147/IJGM.S50243 [Accessed 02.03.2015]

Kumar, K.H., Elavarasi, P., 2016. Definition of pain and classification of pain disorders. *J. Adv. Clin. Res. Insights.*, 3(3), pp.87-90.

Kump, D.S., Booth, F.W., 2005. Alterations in insulin receptor signalling in the rat epitrochlearis muscle upon cessation of voluntary exercise. *J. Physiol*., 562(3), pp.829–838.

Kvæl, L., Bergland, A., Telenius, E. W., 2017. Associations between physical function and depression in nursing home residents with mild and moderate dementia: a cross-sectional study. *BMJ Open*, 7(7), pp.e016875.

Lamé, I.E., Peters, M.L., Kessels, A.G., Van Kleef, M. and Patijn, J., 2008. Test—retest stability of the pain catastrophizing scale and the tampa scale for kinesiophobia in chronic pain over a longer period of time. *J. Health Psychol.*, 13(6), pp.820-826.

Langevin, H.M., Sherman., 2007. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. *Med. Hypotheses*., 68(1), pp.74-80.

Law, L. F., Sluka, K. A., 2017. How does physical activity modulate pain? *Pain*, 158(3), pp.369–370.

Lawford, B.J., Walters, J., Ferrar, K., 2015. Does walking improve disability status, function, or quality of life in adults with chronic low back pain? A systematic review. *Clin. Rehabil*., 30(6), pp.523-536.

Lee, I.M., Djousse, L., Sesso, H.D., Wang, L., Buring, J.E., 2010. Physical activity and weight gain prevention. *JAMA*., 303(12), pp.1173-1179.

Lee, J.S., Kang, S.J., 2016. The effects of strength exercise and walking on lumbar function, pain level, and body composition in chronic back pain patients *J. Exerc. Rehabil.*, 12, pp.463-470.

Leeuw, M., Goossens, M.E., Linton, S.J., Crombez, G., Boersma, K., Vlaeyen, J.W.S., 2007. The Fear-Avoidance Model of Musculoskeletal Pain: Current State of Scientific Evidence. *J. Behav. Med.*, 30(1), pp.77-94.

Legrain, V., Ianetti, G.D., Plaghki, L., Mouraux, A., 2011. The pain matrix reloaded: a salience detection system for the body. *Prog. Neurobiol*., 93(1), pp.111-124.

Li, Y., Dorsi, M.J., Meyer, R.A., Belzberg, A.J., 2000. Mechanical hyperalgesia after an L5 spinal nerve lesion in the rat is not dependant on input from injured nerve fibres. *Pain.*, 85(3), pp.493-502.

Liddle, S.D., Baxter, G.D., Gracey, J.H., 2004. Exercise and chronic low back pain: what works? *Pain*., 107(1-2), pp.176-190.

Liddle, S.D., Baxter, G.D., & Gracey, J.H., 2007. Chronic low back pain: Patients' experiences, opinions and expectations for clinical management. *Disabil. Rehabil*., 29(24), pp.1899-1909

Liddle, S.D., Baxter, G.D., Gracey, J.H., 2009. Physiotherapists' use of advice and exercise for the management of chronic low back pain: a national survey. *Man Ther.*,14(2), pp.189-**1**96.

Lin, C.W., McAuley, J.H., Macedo, l., Barnett, D.C., Smeets, R.J., Verbunt, J.A., 2011. The relationship between physical activity and disability in low back pain: A systematic review and meta-analysis. *Pain*., 152(3), pp.607-613.

Linton, S.J., 2000. A review of psychological risk factors in back and neck pain. *Spine*., 25(9), pp.1148–1156.

Lloyd, D., Findlay, G., Roberts, N., Nurmiko, T., 2008. Differences in low back pain behaviour are reflected in the cerebral response to tactile stimulation of the lower back. *Spine*., 33(12), pp.1372-1377.

Longo, U.G., Loppini, M., Denaro, L., Maffulli, N., Denaro, V., 2010. Rating scales for low back pain, *Brit. Med. Bull.*, 94(1)., pp.81-144.

Louw, Q.A., Morris, L.D., Grimmer-Somers, K., 2007. The prevalence of low back pain in Africa: a systematic review. *BMC Musculoskel. Disord*., 8(1) pp.105.

Louw, A., Diener, I., Butler, D.S., Puentedura, E.J., 2011. The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain. *Arch. Phys. Med. Rehabil*., 92(12), pp.2041-56. Available from: https://doi: 10.1016/j.apmr.2011.07.198. [Accessed 05.08.17]

Louw, A., Zimney, K., Puentedura, E.J., Diener, I., 2016. The efficacy of pain neuroscience education on musculoskeletal pain: A systematic review of the literature. *Physiother Theory Pract*., 32(5), pp.332-55.

Lundberg, M., Grimby-Ekman, A., Verbunt, J., Simmonds, M. J., 2011. Pain-related fear: a critical review of the related measures. [Online] *Pain research and treatment*, *2011*, 494196. Available from: https//: doi:10.1155/2011/494196. [Accessed 20.05.2015]

Magalhães, M.O., Muzi, L.H., Comachio, J., Burke, T.N., França, F.J.R., Ramos, L.A.V., Almeida, G.P.L., Carvalho-e-Silva, A.P.M.C., Marques, A.P., 2015. The short-term effects of graded activity versus physiotherapy in patients with chronic low back pain: a randomized controlled trial. *Man. Ther*. 20, pp.603–609.

Maher, C.G., 2004. Effective physical treatment for chronic low back pain. *Orthop. Clin. N. Am.*, 35(1), pp.57-64.

Maitland, J., 1986. *Vertebral Manipulation*. London: Butterworths

Major-Helsloot, M.E., Crous, LC., Grimmer-Somers, K., Louw, Q.A., 2014. Management of LBP at primary care level in South Africa: up to standards? *Afr. Health Sci*. 14(3), pp.698-706.

Mankovsky, T., Lynch, M.E., Clark, A.J., Sawynok, J., Sullivan, M.J.L., 2012. Pain catastrophising predicts poor response to topical analgesics in patients with neuropathic pain. *Pain Res. Manage*., 17(1), pp.10-14.

Manniche, C., Lundberg, E., Christensen, I., Benzen, L., 1988. Clinical trial of intensive muscle training for chronic lower back pain. *Lancet*, 2, pp.1473-6.

Mannion, A.F., Balagué, F., Pellisé, F. and Cedraschi, C., 2007. Pain measurement in patients with low back pain. *Nat. Rev. Rheumatol.*, *3*(11), pp.610.

Mannion, A.F., Müntener, M., Taimela, S. and Dvorak, J., 1999. Volvo award winner in clinical studies: a randomized clinical trial of three active therapies for chronic low back pain. *Spine*, 24(23), pp.2435.

Marshall, P.W.M, Kennedy, S., Brooks, C., Lonsdale, C. 2013. Pilates exercise or stationary cycling for chronic nonspecific low back pain: does it matter? a randomized controlled trial with 6-month follow-up. *Spine* (Phila Pa 1976) 38:E952–9. Available from: https//: doi:10.1097/BRS.0b013e318297c1e5 [Accessed 12.06.2017]

Martinez-Calderon, J., Flores-Cortes, M., Morales-Asencio, J.M., Luque-Suarez, A,. 2020. Conservative Interventions Reduce Fear in Individuals With Chronic Low Back Pain: A Systematic Review. *Arch Phys Med Rehabil*., 101(2), pp.329-358.

Martinez, C.H., Moy, M.L., Nguyen, H.Q., Cohen, M., Kadri, R., Roman, P., Holeman, R.G., Kim, H.M., Goodrich, D.E., Giardino, N.D., Richardson, C.R., 2014. Taking healthy steps: rationale, design and baseline characteristics of a randomized trial of a pedometer-based internet-mediated walking program in veterans with chronic obstructive pulmonary disease. *BMC Pulm. Med*., 14(1), pp.12.

Matchaya, M., Muula, A.S., 2009. Perceptions towards private medical practitioners' attachments for undergraduate medical students in Malawi. *Malawi Med J*., 21(1), pp.6-11.

Mathieson, S., Lin, C., 2013. Appraisal. painDETECT questionnaire. *J. Physiother.*, 59(3), pp.211.

Mayer, J., Mooney, V., Dagenais, S., 2008. Evidence-informed management of chronic low back pain with lumbar extensor strengthening exercises. *Spine Journal.*, 8(1), pp.96-113.

Mayo, T.P., Weissman, L., 2011. The noninvasive path to chronic back pain management. *Rehab Manag. Interdiscip. J. Rehabil*. 24, pp.18-20.

McCurry, S. M., Pike, K. C., Vitiello, M. V., Logsdon, R. G., Larson, E. B., Teri, L., 2011. Increasing walking and bright light exposure to improve sleep in community-dwelling persons with Alzheimer's disease: results of a randomized, controlled trial. *JAGS*., 59(8), pp.1393–1402.

McDonough, S.M., Tully, M.A., Boyd, A., O'Connor, S.R., Kerr, D.P., O'Neill, S.M.O., Delitto, A., Bradbury, I., Tudor-Locke, C., Baxter, G.D., Hurley, D.A., 2013. Pedometer driven walking for chronic low back pain. A Feasibility Randomized Controlled Trial. *Clin. J. Pain*., 29(11), pp.972-981.

McKenzie, R., 2000. Mechanical diagnosis and therapy for disorders of the low back. In: T. Taylor ed. *Physical Therapy for the Low Back.* New York: Churchill Livingstone.pp.141-66.

McMahon, S.B., Bennet, D.L.H., Bevan, S., 2006. Inflammatory mediators and modulators of pain. In: McMahon, S.B., Koltzenburg, M., eds. *Wall and Melzack's Textbook of Pain*. 5th ed. London: Elsevier, pp.40-72.

Meagher, M.W., Arnau, R.C., Rhudy, J.L., 2001. Pain and Emotion: Effects of Affective Picture Modulation. *Psychosom. Med*., 63(1), pp.79-90.

Melzack, R., Wall, P., 1965. Pain Mechanisms: A New Theory. *Science.*, 150 (3699), pp.971-979.

Merom, D., Rissel, C., Phongsavan, P., Smith, B.J., Van Kemenade, C., Brown, W.J., Bauman, A.E., 2007. Promoting walking with pedometers in the community: the step-by-step trial. *Am. J. Prev. Med*., 32(4), pp.290-297.

Meucci, R.D., Fassa, A.G., Faria, N.M.X., 2015. Prevalence of chronic low back pain: a systematic review. [Online] *Rev Sau'de Publica*.,49(73) Available from: https//: doi:10.1590/S0034-8910.2015049005874 [Accessed: 05:04.2017]

Meyer, H.P., 2007. Pain management in primary care – current perspectives. *SA Fam. Pract*., 49(7), pp.20-25.

Meyer, R.A., Ringkamp, M., Campbell, J.N., Raja, S.N., 2006. Peripheral mechanisms of cutaneous nociception. In: McMahon, S.B., Koltzebburg, eds. *Wall and Melzack's Textbook of Pain*. 5th ed. London: Elsevier, pp.3-34.

Micklesfield, L.K., Lambert, E.V., Hume, D.J., Chantler,, S., Pienaar, P.R., Dickie, K., Puoane, T., Goedecke, J.H., 2013. Socio-cultural, environmental and behavioural determinants of obesity in black South African women. *Cardiovasc J Afr*., 24(9-10), pp.369-375.

Miller, R.P., Kori, S.H., Todd, D.D., 1991. The Tampa Scale: a measure of kinesiophobia. *Clin J Pain.*, 7, pp.51–52.

Miller, J.S., Pinnington, M.A., Stanley, I.M., 1999. The early stages of low back pain: a pilot study of patient diaries as a source of data. *Fam. Pract*., 16(4), pp.395-401.

Milosavljevic, S., Clay, L., Bath, B., Trask, C., Penz, E., Stewart, S., Hendrick, P., Baxter, G.D., Hurley, D.A., McDonough, S.M., 2015. Walking away from back pain: one step at a time- a community-based randomised controlled trial. *BMC Public Health*. 15(1), pp.144.

Mirovsky, Y., Grober, A., Blankstein. A., Stabholz, L., 2006. The effect of ambulatory traction combined with treadmill on patients with chronic low back pain. *J. Back Musculoskel. Rehabil*., 19(2-3), pp.73-78.

Moher, D., Hopewell, S., Shulz, K.F., Motori V., Gotzsche, P.C., Devereaux, P.J., Elbourne, D., Egger, M., Altman, D.G., 2010. Consort 2010 Explanation and Elaboration: updated guidelines for reporting parallel group randomised trials.[Online*] J. Clin. Epidemiol*.,63, pp.e1-e37. Available from: www.ac.els-cdn.com.ezp2.bath.ac.uk [Accessed 29.05.2015]

Monninkhof, E.M., Elias, S.G., Vlems F.A., van der Tweel, I., Schuit, A.J., Voskuil, D.W., van Leeuwen, F.E., 2007. Physical activity and breast cancer: a systematic review. *Epidemiol*., 18(1), pp.137-157.

Moore, J.E., 2010. Chronic low back pain and psychosocial issues. *Phys. Med. Rehabil. Clin. N. Am*., 21(4), pp.801-815.

Moore, R.A., McQuay, H.J., 2005. Prevalence of opioid adverse events in chronic non-malignant pain: a systematic review of randomised trials of oral opioids. [Online] *Arthritis Res. Ther*., 7(5), pp. R1046-51. Available from: https://arthritis-research.com/content17/5/R1046 [Accessed 02.03.2015]

Moore, R.A., Derry, S., Wiffen, P.J., 2013. Challenges in design and interpretation of chronic pain trials. *Br J Anaesth*., 111(1), pp.38-45.

Moore, S.C., Patel, A.V., Matthes, C.E., Berrington de Gonzalez, A., Park, Y.,KAtki, H.A., Linet, M.S., Weiderpass, E., Visvanathan, K., Helzlsouer, K.J.,Thun, M., Gapstur, S.M., Hartge, P., Lee, I.M., 2012. Leisure time physical activity of moderate to vigorous intensity and mortality: A large pooled cohort analysis [Online]. *PLoS Med*., 9(11), pp.e1001335. Available from: https://doi.10.1371/journal.pmed.1001335 Accessed on: [02.02.2017]

Mora, S., Cook, N., Buring, J.E., Ridker, P.M., Lee, I.M., 2007. Physical activity and reduced risk of cardiovascular events: potential mediating mechanisms. *Circulation.,* 116(19), pp.2110–2118.

Morlion, B., 2013. Chronic low back pain: Pharmacological, interventional and surgical strategies. *Nat Rev Neurol*., 9(8), pp.462-473.

Morris, L.D., Grimmer-Somers, K.A., Louw, Q.A., Sullivan, M.J., 2012. Cross-cultural adaptation and validation of the South African Pain Catastrophising Scale (SA-PCS) among patients with fibromyalgia. *BMC. Health Qual. Life Outcomes.,* 10(1) pp.137.

Morris, L. D., Daniels, K. J., Ganguli, B., & Louw, Q. A. 2018. An update on the prevalence of low back pain in Africa: a systematic review and meta-analyses. *BMC musculoskeletal disorders*, *19*(1), 196. Avaialble from: https://doi:10.1186/s12891-018-2075-x Accessed on : [05.02.2019]

Morrison, I., Perini, I., & Dunham, J. 2013. Facets and mechanisms of adaptive pain behavior: predictive regulation and action. *Frontiers in human neuroscience*., *7,* 755. Available from: https: doi:10.3389/fnhum.2013.00755. [Accessed: 03.05.2018]

Moseley, L., 2002. Combined physiotherapy and education is efficacious for chronic low back pain. *Australian Journal of Physiotherapy.,* 48(4), pp.297-302.

Moseley, L., 2003. Unravelling the Barriers to Reconceptualization of the Problem in Chronic Pain: The Actual and Perceived Ability of Patients and Health Professionals to Understand the Neurophysiology. *J. Pain*., 4(4), pp.184-189.

Moseley, G.L., Arntz, A., 2007. The context of a noxious stimulus affects the pain it evokes. *Pain.*, 133(1-3), pp.64-71.

Murphy, M.H., Blair, S.N., Murtagh, E.M., 2009. Accumulated versus continuous exercise for health benefit: a review of empirical studies. *Sports Med.*, 39(1), pp.29-43.

Nachemson, A., 1999. Back Pain: delimiting the problem in the next millennium. *Int. J. Law Psychiatry*, 22(5-6), pp.473-480.

Nagai, M., Kuriyama, S., Kakizaki, M., Ohmori-Matsuda, K., Sone, T., Hozawa, A., Kawado, M., Hashimoto, S., Tsuji, I., 2011. Impact of walking on life expectancy and lifetime medical expenditure: the Ohsaki Cohort Study. *BMJ open*, 1(2), pp.e000240. Available from: https//: doi:10.1136/bmjopen-2011-000240 [Accessed 21.02.2017]

Naidoo, V., Mudzi, W., Ntsiea, V., Becker, P.J., 2012. Physiotherapy Modalities used in the Management of Chronic Low Back Pain. *SA J. Physiother*., 68(1), pp.42-46.

Narasimooloo, C., 2011. Adequacy of pain management in HIV-positive patients. *SA Fam. Pract*., 53(1), pp.71-76.

National Institute for Health Research, 2019. *Moving Matters - Interventions To Increase Physical Activity*.[Online] Available from: https://www.dc.nihr.ac.uk/themed-reviews/research-into-physicalactivity.htm?utm_source=NIHR+Dissemination+Centre+mailing+list&utm_campaign=cce20eb83d-EMAIL_CAMPAIGN_2019_07_01_08_52&utm_medium=email&utm_term=0_286155606c-cce20eb83d-167853685. [Accessed 03.07.2019]

National Institute for Health and Care Excellence. 2016.Low back pain and sciatica in over 16s:assessment and management .[Online] London: NICE Available from: https://www.nice.org.uk/guidance/ng59/chapter/Recommendations [Accessed 05.04.2019]

National Institute for Health and Care Excellence. 2019. Neuropathic pain in adults: pharmacological management in non-specialist settings.[Online] London: NICE Available from: https://www.nice.org.uk/guidance/cg173 [Accessed 05.04.2019]

National Collaborating Centre for Primary Care (UK), 2009. *Low Back Pain: Early Management of Persistent Non-spesific Low Back Pain NICE Clinical Guidelines, No. 88* [Online] London: Royal College of General Practitioners. Available from: https://www.ncbi.nlm.nih.gov.books/NBK11702/?report=reader#po=25.0000 [Accessed 14. 02. 2014]

Nicholas, M.K., Asghari, A., Corbett, M., Smeets, R.J.E.M., Wood, B.M., Overton, S., Perry, C., Tonkin, L.E., Beeston, L., 2012. Is adherence to pain self-management strategies associated with improved pain, depression and disability in those with disabling chronic pain? *Eur. J. Pain*., 16(1), pp.93-104.

Nieman, D.C., Henson, D.A., Austin, M.D., Brown, V.A., 2005. Immune response to a 30-minute walk. Randomized controlled trial. *Med. Sci. Sports Exerc.*, 37(1), pp.57-62.

Nijs, J., Apeldoorn, A., Hallegraeff, H., Clark, J., Smeets, R., Malfliet, A., Girbes, E.L., De Koning, M., Ickmans, K., 2015. Low Back Pain: guidelines for the clinical classification of predominant neuropathic, nociceptive, or central sensitization pain. *Pain Physician*., 18(3), pp.E333-346.

Nijs, J., Roussel N., Paul van Wilgen, C., Köke, A., Smeets, R. 2013. Thinking beyond muscles and joints: therapists' and patients' attitudes and beliefs regarding chronic musculoskeletal pain are key to applying effective treatment. *Man. Ther.*, 18(2), pp.96–102.

Ogon, M., Krismer, M., Söllner, W., Kantner-Rumplmair, W. and Lampe, A., 1996. Chronic low back pain measurement with visual analogue scales in different settings. *Pain.*, 64(3), pp.425-428.

Ogunlana, M.O., Odole, A.C., Adejumo, A., Olagbegi, O.M., Williams, O.O., 2018. Augmenting conventional treatment of non-specific low back pain with progressive goal attainment programme, *Phys. Ther. Rev.*, 23(2). pp.133-143.

Omokhodion, F.O., Sanya, A.O., 2003. Risk factors for low back pain among office workers in Idadan, Southwest Nigeria. *Occup. Med*., 53(4), pp.287-289.

Omron ® Healthcare, INC, 2012. Omron ® Instruction Manual Pedometre Model: HJ-123. [Online] New York: Omron® Healthcare, INC. Avaible from: https://www.omronhealthcare.com/wp-content/uploads/HJ-321-IM-WEB-03212012.pdf [Accessed 12.05.2019]

Omura, J.D., Ussery, E.N., Loustalot, F., Fulton, J.E., Carlson, S.A., 2019. Walking as an opportunity for cardiovascular disease prevention. [Online] *Prev Chronic Dis*., Available from: https://doi.10.5888/pcd16.180690 [Accessed 03.03.2019]

Orozco, T., Feldman, D.E., Mazer, B., Chilingaryan, G., Hunt, M., Williams-Jones, B., Laliberté, M., 2017. Low Back Pain: Current Patterns of Canadian Physiotherapy Service Delivery. *Physiother Can*., 69(1), pp.49-56.

Oruç, Z., Kaplan, M. A., 2019. Effect of exercise on colorectal cancer prevention and treatment. *World J. Gastrointest. Oncol*., 11(5), pp.348–366.

Ossipov, M.H., Dussor, G.O., Porreca, F., 2010. Central modulation of pain. *J. Clin. Invest*., 120(11), pp.3779-3787.

Osman, A., Barrios, F.X., Kopper, B.A., Hauptmann, W., Jones, J. and O'Neill, E., 1997. Factor structure, reliability, and validity of the Pain Catastrophizing Scale. *J. Behave. Med.*, 20(6), pp.589-605.

Osman, A., Barrios, F.X., Gutierrez, P.M., Kopper, B.A., Merrifield, T. and Grittmann, L., 2000. The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. *J. Behav. Med.*, 23(4), pp.351-365.

Ostelo, R.W.J.G., de Wet, H.C.W., 2005. Clinically important outcomes in low back pain. *Best Pract. Res. Clin. Rheumatol*., 19(4), pp.593-607.

Ostelo, R.W., Deyo, R.A., Stratford, P., Waddell, G., Croft, P., Von Korff, M., Bouter, L.M. and Henrica, C., 2008. Interpreting change scores for pain and functional status in low back pain: towards international consensus regarding minimal important change. *Spine.*, 33(1), pp.90-94.

Osterweis, M., Kleinman, A. and Mechanic, D.,eds. 1987. *The Anatomy and Physiology of Pain*. In: *Pain and Disability: Clinical, Behavioral, and Public Policy Perspectives*. Washington (DC): National Academies Press (US)

O'Sullivan, P., Twomey, L., Allison, G., 1997. Dysfunction of the neuromuscular system in the presence of low back pain – implications for physical therapy management. *J. Man. Manip. Ther*., 5(1), pp.20-26.

O' Sullivan, P., 2005. Diagnosis and classification of chronic low back pain disorders: maladaptive movement and motor control impairments as underlying mechanism. *Man Ther*., 10(4), pp.242-255.

Ouédraogo, D.D., Nonguierma, V., Napon, C., Kabré, A., Tiéno, H., Guira, O., Kaboré, J., Drabo, J.Y., 2012. Prevalence of neuropathic pain amongst Black African patients suffering from common low back pain. *Rheumatol. Int*., 32(7), pp.2149-2153.

Page, P., 2014. Beyond statistical significance: clinical interpretation of rehabilitation research literature. *International journal of sports physical therapy*, *9*(5), pp.726–736.

Pan, H.L., Eisenach, J.C., Chen, S.R., 1999. Gabapentin suppresses ectopic nerve discharges and reverses allodynia in neuropathic rats. *J. Pharmacol. Exp. Ther*., 288(3), pp.1026-1030.

Patel, S.M., Stason, W.B., Legedza, A., Ock, S.M., Kaptchuk, T.J., Conboy, L., Canenguez, K., Park, J.K., Kelly, E., Jacobson, E., Kerr, C.E., Lembo, A.J., 2005. The placebo effect in irritable bowel syndrome trials: a meta-analysis. *Neurogastroenterol Motil*., 17(3), pp.332-340.

Parent, A., 1996. *Carpenter's Human Neuroanatomy*. 9th ed. London: Williams & Wilkins.

Parker, R., Bergman, E., Mntambo, A., Stubbs, S., Wills, M., 2017. Levels of physical activity in people with chronic pain [Online], *SA J. Physiother*., 73(1), pp.a323. Available from: https://doi.org/10.4102/sajp.v73i1.323 [Accessed 25.04.2018]

Parr, S., May, S., 2014. Do musculoskeletal physiotherapists believe the NICE guidelines for the management of non-specific LBP are practical and relevant to their practice? : a cross sectional survey. *Physiother.*, 100(3), pp.235-241.

Pederson, B.K., Saltin, B., 2015. Exercise as medicine – evidence for prescribing exercise as therapy in 26 different chronic diseases. *Scand J Med Sci Sports.*, 25(3), pp.1-72.

Peduzzi, P., Concato, J., Kemper, E., Holford, T.R., Feinstein, A.R., 1996. A simulation study of the number of events per variable in logistic regression analysis. *J. Clin. Epidem.*, 49(12), pp.1373-1379.

Pereira, F.G., França, M.H., Paiva, M.C.A.D., Andrade, L.H. and Viana, M.C., 2017. Prevalence and clinical profile of chronic pain and its association with mental disorders. *Revista de saude publica*, 51 p.96.

Pergolizzi, J., Böger, R.H., Budd, K., Dahan, A., Erdine, S., Hans, G., Kress, H.G., Langford, R., Likar, R., Raffa, R.B., Sacerdote, P., 2008. Opiods and the management of chronic severe pain in the elderly: consensus statement of an International Expert Panel with focus on the six clinically most used World Health Organization Step III opioids (budrenorphine, fenatyl, hydromorphone, methadone, morphine, oxycodone). *Pain Pract.*, 8(4), pp.287-313.

Peters, M.L., Vlaeyen, J.W.S., Weber, W.E.J., 2005.The joint contribution of physical pathology, pain-related fear and catastrophizing to chronic back pain disability. *Pain.,* 113(1-2), pp.45-50.

Picavet, H.S., Vlaeyen, J.W., Schouten, J.S., 2002. Pain catastrophising and kinesiophobia: predictors of chronic low back pain. *Am. J. Epidemiol*., 156(11), pp.1028-1034.

Picelli, A., Buzzi, M.G., Cisari, C., Gandolfi, M., Porru, D., Bonadiman, S., Brugnera, A., Carone, R., Cerbo, R., Del Carro, U., Gimigliano, R., Invernizzi, M., Miotto, D., Nappi, R., Negrini, S., Scheiger, Picelli, A., Buzzi, M.G., Cisari, C., Gandolfi, M., Porru, D., Bonadiman, S., Brugerna, A., Carone, R., Cerbo, R., Del, U.C., Gimigliano, R., 2016. Headache, low back pain, other nociceptive and mixed pain conditions in neurorehabilitation. Evidence and recommendations from the Italian Consensus Conference on Pain in Neurorehabilitation. *Eur. J. Phys. Rehabil. Med*., 52(6), pp.867-880.

Pillay, J. D., van der Ploeg, H. P., Kolbe-Alexander, T. L., Proper, K. I., van Stralen, M., Tomaz, S. A., van Mechelen , W., Lambert, E. V., 2015. The association between daily steps and health, and the mediating role of body composition: a pedometer-based, cross-sectional study in an employed South African population. *BMC Public Health.*, *15*, 174. Available from: https://doi:10.1186/s12889-015-1381-6. [Accessed 22.03.2017]

Pincus, T., Smeets, R.J.E.M., Simmonds, M.J., Sullivan, M.J.L., 2010. The Fear Avoidance Model Disentangled: Improving the Clinical Utility of the Fear Avoidance Model. *Clin. J. Pain*., 26(9), pp.739-746.

Pinto, R.Z., Ferreira, M.L., Oliveira, V.C., Franco, M.R., Adams, R., Maher, C.G., Ferreira, P.H., 2012. Patient-centred communication is associated with positive therapeutic alliance: a systematic review. J *Physiother*., 58(2), pp.77-87.

Preyde, M., 2000. Effectiveness of massage therapy for subacute low-back pain: a randomized controlled trial. *CMAJ*., 162(13), pp.1815-1820.

Privett, T., 2012. Aerobic exercise as a means of reducing low back pain a systematic review. [Online]. Available from: https://stars.library.ucf.edu/honorstheses1990-2015/1293 [Accessed 11.02.2016]

Potter, J., Higginson, I. J., Scadding, J. W., & Quigley, C., 2003. Identifying neuropathic pain in patients with head and neck cancer: use of the Leeds Assessment of Neuropathic Symptoms and Signs Scale. *J. Royal Soc. Med.*, 96(8), pp. 379–383.

Pope, M.H., Philips, R.B., Haugh, L.D., Hsieh, C.Y., MacDonald, L., Haldeman, S., 1994. A prospective randomized three-week trial of spinal manipulation, transcutaneous muscle stimulation, massage and corset in the treatment of subacute low back pain. *Spine*., 19(22), pp.2571-2577.

Public Health England. 2018. Active 10 – Because There's Only One You. [Online] UK: National Health Service Available from: https://www.nhs.uk/oneyou/active10/home [Accessed 15.10. 2018].

Puentedura, E.J., Louw, A., 2012. A neuroscience approach to managing athletes with low back pain. *Phys Ther Sport.*, 13(3), pp.123-33.

Puentedura, E.J., Flynn, T., 2016. Combining manual therapy with pain neuroscience education in the treatment of chronic low back pain: A narrative review of the literature. *Physiother Theory Pract*., 32(5), pp.408-414.

Purves, D., Augustine, G.J., Fitzpatrick, D., LaMantia, A., Williams, S.M., 2001. *Neuroscience.* 2nd ed. Sunderland, Massachusetts: Sinauer Associates.

Qaseem, A., Wilt, T.J., McLean, R.M., Forciea, M.A., 2017. Clinical Guidelines Committee of the American College of Physicians. Noninvasive Treatments for Acute, Subacute, and Chronic Low Back Pain: A Clinical Practice Guideline From the American College of Physicians. *Ann Intern Med*.,166(7), pp.514–530

Quartana, P.J., Campbell, C.M., Edwards, R.R., 2009. Pain catastrophising: a critical review. *Expert Rev Neurother.*,9(5), pp.745-758.

Rainville, J., Hartigan, C., Martinez, E., Limke, J., Jouve, C., Finno, M., 2004. Exercise as a treatment for low back pain. *Spine.*, 4(1), pp.106-105.

Ramer, M.S., Thompson, S.W., McMahon, S.B., 1999. Causes and consequences of sympathetic basket formation in dorsal root ganglia. *Pain*., 82(Supplement), pp.S111-120.

Rantanen, P., 2001. Physical measurements and questionnaires as diagnostic tools in chronic low back pain. *J. Rehabil. Med.*, 33(1), pp.31-35.

Rapaport, M.H., Schettler, P., Bresee, C., 2012. "A preliminary study of the effects of repeated massage on hypothalamic-pituitary- adrenal and immune function in healthy individuals: a study of mechanisms of action and dosage," *J. Alter. Complement. Med*., 18(8), pp.789–797.

Raymond, A., Bouton, C., Richard, I., Roquelaure, Yves, Baufreton, C., Legrnd, E., Huez, J.F., 2011. Psychosocial risk factors for chronic low back pain inprimary care – a systematic review.[Online] *Fam Pract,*. 28(1), pp.12-21.

Rehm, S., Koroschetz, J., Baron, R., 2008. An Update on Neuropathic Pain. *Eur. Neurol. Rev*., 3(1), pp.125-127.

Reicherts, P., Gerdes, A.B.M.,Pauli, P., Wieser, M.J., 2016. Psychological Placebo and Nocebo Effects on Pain Rely on Expectation and Previous Experience. *J. Pain*., 17(2), pp.1-12.

Rezende, L.F.M, de Sa', T.H., Markozannes, G., Rey-Lopez, J.P., Lee, I.M., Tsilidis, K.K., Ioannidis, J.P.A., 2018. Physical activity and cancer: an umbrella review of the literature including 22 major anatomical sites and 770 000 cancer cases. *Br J. Sports Med*., 52(13), pp.826-833.

Richardson, C., Hodges, P.W., Hides, J. 2004. *Therapeutic Exercise for Lumbopelvic Stabilization: A Motor Control Approach for the Treatment and Prevention of Low Back Pain*. 2nd ed. Edinburgh: Churchill Livingstone.

Richardson, C.R., Newton, T.L., Abraham, J.J., Sen, A., Jimbo, M., Swartz, A.M., 2008. A Meta-Analysis of Pedometer-Based Walking Interventions and Weight Loss. *Ann. Fam. Med*., 6(1), pp.69-77.

Rodriguez-Sanchez, E., Criado-Gutierrez, J.M., Mora-Simon, S., Muriel-Diaz, M.P., Gomez-Marcos, M.A., Recio-Rodriguez, J.I., Patino-Alonso, M.C., Valero-Juan, L.F., Maderuelo-Fernandez, J.A., Carcia-Ortiz, L., DERIVA Group., 2014. Physical activity program for patients with dementia and their relative caregivers: randomized clinical trial in Primary Health Care (AFISDEMyF study). [Online] *BMC Neurol*., 14(63). Available from: https://doi.org/10.1186/1471-2377-14-63 [Accessed 05.02.2015]

Roelofs, J., van Breukelen, G., Sluiter, J., Frings-Dresen, M.H.W., Goosens, M., Thibault, P., Boersma, K., Vlaeyen, J.W.S., 2011. Norming of the Tampa Scale for Kinesiophobia across pain diagnoses and various countries. *Pain*, 152, pp.1090-1095.

Roland, M. and Fairbank, J., 2000. The Roland–Morris disability questionnaire and the Oswestry disability questionnaire. *Spine*, 25(24), pp.3115-3124.

Roland, M., 2002. The back book: the best way to deal with back pain: get back active. Norwich: The Stationary Office, 2002.

Romanò, C.L., Romanò, D., Lacerenza, M., 2012. Antineuropathic and Antinociceptive Drugs Combination in Patients with Chronic Low Back Pain: A Systematic Review. [Online] *Pain Res. Treat*., 12(1) ID 154781, 1-8. Available from: http://dx.doi.org/10.1155/2012/154781 [Accessed 02.03.2015]

Roussel, N.A., Nijs, J., Meeus, M., Mylius, V., Fayt, C., Oostendorp, R., 2013. Central sensitization and altered central pain processing in chronic low back pain: Fact or myth? *Clin. J. Pain.*, 29(7), pp.625-638.

Rowbothom, M., Harden, N., Stacey, B., Bernstein, P., Magnus-Miller, L., 1998. Gabapentin for the treatment of postherpetic neuralgia: a randomized controlled trial. *JAMA*. 280(21), pp.1837-1842.

Rubin, D, I., 2007. Epidemiology and Risk factors for Spine Pain. *Neurol. Clin.*, 25(2). pp.353-371.

Rubinstein, S.M., van Middelkoop, M., Assendelft, W.J.J., de Boer, M.R., van Tulder, M.W., 2011. Spinal manipulative therapy for chronic low-back pain. Cochrane Database of Systematic Reviews, Issue 2. Art. No.: CD008112. Available from: https: DOI: 10.1002/14651858.CD008112.pub2 [Accessed 11.03.2016]

Ruddock, J. K., Sallis, H., Ness, A., Perry, R. E., 2016. Spinal Manipulation Vs Sham Manipulation for Nonspecific Low Back Pain: A Systematic Review and Meta-analysis. *J. Chiropr. Med.*, *15*(3), pp.165-183.

Ryan, C.G., Grant, P.M., Dall, P.M., Gray, H., Newton, M., Granat, M.H., 2009. Individuals with chroniclow back pain hav e alowerlevel, and an altered pattern, of physical activity compared with matched controls: an observational study. *Aus. J. Physiother*., 55(1), pp.53-58.

Saltychev, M., Mattie, R., McCormick, Z., Bärlund, E. and Laimi, K., 2017. Psychometric properties of the oswestry disability index. *Int. J. Rehabil. Res.*, 40(3), pp.202-208.

Sahrmann, S., Azevedo, D. C., & Dillen, L. V., 2017. Diagnosis and treatment of movement system impairment syndromes. *Brazilian journal of physical therapy, 21*(6), pp.391–399.

Ŝarabon, N. 2011. Effects of trunk functional stability training in subjects suffering from chronic low back pain: A pilot study. *Kinesiol. Slov*. 17(2), pp.25-37.

Sanderson, T., Morris, M., Calnan, M., Richards, P., Hewlett, S., 2010. Patient perspective of measuring treatment efficacy: the rheumatoid arthritis patient priorities for pharmacologic interventions outcomes. *Arthritis Care Res.*, 62(5), pp.647-656.

Savvaa, C., Giakasb, G., Efstathioua, M., 2014. The role of the descending inhibitory pain mechanism in musculoskeletal pain following high-velocity, low amplitude thrust manipulation. A review of the literature. J. *Back Musculoskel. Rehabil*., 27(4), pp.377-382.

Schäfer, A., Hall, T., Briffa, K., 2009. Classification of low back-related leg pain- A proposed patho-mechanism-based approach. *Man. Ther*., 14(2), pp.222-230.

Schaller, A., Dejonghe, L., Haastert, B., Froboese, I., 2015. Physical activity and health-related quality of life in chronic low back pain patients: a cross-sectional study. [Online] *BMC Musculoskel. Disord.*, 16(62). Available from: https: doi:10.1186/s12891-015-0527-0 [Accessed 15.09.2017]

Scholz, J., Mannion, R.J., Hord, D.E., Griffin, R.S., Rawal, B., Zheng, H., Scoffings, D., Phillips, A.,Guo, J.,Laing, R.J.C., Abdi, S., Decosterd, I., Woolf, C.J., 2009. A Novel tool for the assessment of pain: Validation in low back pain. [Online] *PLOS Med*., 6(4), pp.e1000047. Available from: https://doi.org/10.1371/journal.pmed.1000047 [Accessed on 10.12.2012].

Schulz, K.F., Altman, D.G., Moher, D., 2010. CONSORT 2010 Statement: updated guidelines for reporting parallel group randomised trials. *Trials* 11, 32 (2010). Available from: https://doi.org/10.1186/1745-6215-11-32 [Accessed on 03.12.2016].

Schűtze, R., Rees, C., Smith, A., Slater, H., Campbell, J.M., O'Sullivan, P., 2017. How can we best reduce Pain Catastrophizing in Adults with Chronic Noncancer Pain? A systematic review and Meta-Analysis. [Online] *The Journal of Pain.*, 19(3). Available from: https: doi/org/10.1016/j.pain.2017.09.010 [Accessed 05.06.2018]

Schweizerhof, M., Stösser, S., Kurejova, M., Njoo, C., Gangharan, V., Agarwal, N., Schmelz, M., Bali, K.K., Michalski, C.W., Brugger, S., Dickenson, A., Simone, D.A., Kuner, R., 2009. Hematopoietic colony-stimulating factors mediate tumor-nerve interactions and bone cancer pain. *Nat. Med*., 15(1), pp.802-807.

Schwellnus, M.P.,Patel, D.N., Nossel, C., Whitesman, S., Derman, E.W., 2011. Healthy Lifestyle interventions in general practice: Part 15: Lifestyle and lower back pain. *SA Fam. Pract*., 53(4), pp.304-311.

Searle, A., Spink, M., Ho, A., Chuter, V., 2015. Exercise interventions for the treatment of chronic low back pain: a systematic review and meta-analysis of randomised controlled trials. *Clin. Rehabil.*, 29(12), pp.1155-1167.

Seminowicz, D.A., Davis, K.D., 2006. Cortical responses to pain in healthy individuals depends on pain catastrophising. *Pain.*, 120(3), pp.297-306.

Senkowski, D., Heinz, A., 2016. Chronic pain and distorted body image: Implications for multisensory feedback interventions. [Online] *Neurosci Biobehav Rev*., 69, pp.252-9. Available from: https: doi: 10.1016/j.neubiorev.2016.08.009. [Accessed 2017.09.20]

Senkowski, D., Hofle, M., Andreas, K.E., 2014. Crossmodal shaping of pain: a multisensory approach to nociception. *Rev. Trends in Cog. Sci*., 18(6), pp.319-327.

Shaygan, M., Böger, A., Kröner-Herwig, B., 2013. Clinical features of chronic pain with neuropathic characteristics: A symptom-based assessment using the Pain DETECT Questionnaire. *Eur J. Pain*. 17(10), pp. 1529-1538.

Shiri, R., Karppinen, J., Leino-Arjas, P., Solovieva, S., Viikari-Juntura, E., 2010. The association between obesity and low back pain: a meta-analysis. *Am J Epidemiol*., 171(2), pp.135-154.

Shnayderman, I., Katz-Leurer, M., 2013. An aerobic walking program versus muscle strengthening programme for chronic low back pain: A randomized controlled trial. *Clin Rehabil*. 27(3), pp.207-214.

Siddall, P.J., Cousins, M.J., Otte, A., Griesing, T., Chambers, R., Murphy, T.K., 2006. Pregabalin in central neuropathic pain associated with spinal cord injury: a placebo-controlled trial. *Neurol*., 67(10), pp.1792-1800.

Siegel, A., Sapru, H.N., 2011, *Essential Neuroscience.* 2nd ed. Philadelphia, Wolters Kluwer Health/ Lippincott Williams & Wilkins.

Sitthipornvorakul, E., Klinsophon, T., Sihawong, R., Janwantanakul, P., 2018. The effects of walking intervention in patients with chronic low back pain: A meta-analysis of randomized controlled trials. *Musculoskelet Sci Pract*., 3, pp.38-46.

Slade, S.C., Molloy, E., Keating, J.L., 2009. People with non-specific chronic low back pain who have participated in exercise programs have preferences about exercise: a qualitative study. *Aust. J. Physiother*., 55(2), pp.115-1121.

Slenz, C.A., Duscha, B.D., Johnson, J.L., Ketchum, K., Aiken, L.B., Samsa, G.P., Houmard, J.A., Bales, C.W., Krause, W.E., 2004. Effects of the amount of exercise on body weight, body composition, and measures of central obesity: STRIDE—a randomized controlled study. *Arch. Intern. Med*., 164(1), pp.31-39.

Sluka, K.A., Law, L.F., Bemet, M.H., 2018. Exercise-Induced pain and analgesia? Underlying mechanisms and clinical translation. Pain.,159 (Suppl 1): S91-S97.Available from: https://doi.org.10.1097/j.pain.0000000000001235. [Accessed 15.05.2017]

Smart, K.M., Blake, C., Staines, A., Thacker, M., Doody, C., 2012a. Mechanism-based classifications of musculoskeletal pain: Part 1 of 3: Symptoms and signs of central sensitization in patients with low back (±) leg. *Man. Ther*., 17(4), pp.336-344.

Smart, K.M., Blake, C., Staines, A., Thacker, M., Doody, C., 2012b. Mechanism-based classifications of musculoskeletal pain: Part 2 of 3: Symptoms and signs of peripheral neuropathic pain in patients with low back (±) leg. *Man.Ther*., 17(4), pp.345-351.

Smart, K.M., Blake, C., Staines, A., Thacker, M., Doody, C., 2012c. Mechanism-based classifications of musculoskeletal pain: Part 2 of 3: Symptoms and signs of nociceptive pain in patients with low back (±) leg. *Man. Ther*., 17(4), pp.352-357.

Smart, K.M., O'Connell, N.E., Doody, C., 2008. Towards a mechanism-based classification of pain in musculoskeletal physiotherapy? *Phys. Ther. Rev*., 13(1), pp.1-10.

Smeets J.R., Vlaeyen J.W.S., Hidding A., Kester A.D.M., Van Der Heijden G.J.M.G., Van Geel A.C.M., Knottnerus J.A., 2006a. Active rehabilitation for chronic low back pain: Cognitive-behavioural, physical or both? First direct post-treatment results from a randomised control trial. [Online] *BMC Musculoskel. Disord*., 7(5), Available from: http//doi.10.1186/1471-2474-7-5 [Accessed 11.02.2014]

Smeets, R.J., Vlaeyen, J.W., Kester, A.D., Knottnerus, J.A., 2006b. Reduction of pain catastrophising mediates the outcome of both physical and cognitive-behavioural treatment in chronic low back pain. *Pain*.,7(4), pp.261-271.

Smeets, R.J.E.M., Wade, D., Hidding, A., van Leeuwen, P.J.C.M., Vlaeyen, J.W.S., Knottnerus., 2006c. The association of physical deconditioning and chronic low back pain: A hypothesis-orientated system. *Disabil. Rehabil*., 28(11), pp.673-693.

Smith, B. E., Littlewood, C.,May, S., 2014. An update of stabilisation exercises for low back pain: a systematic review with meta-analysis. [Online] *BMC musculoskel disord.*, Available from: https//: doi:10.1186/1471-2474-15-416. [Accessed 22.06.2016]

Smith, B.H, Torrance, N., Ferguson, J.A., Bennet, M.I., Serpell, M.G., Dunn, K.M., 2012. Towards a definition of refractory neuropathic pain for epidemiological research. An international Delphi survey of experts. [Online] *BMC Neurol*. 12(29). Available from: https://doi.org/10.1186/1471-2377-12-29 [Accessed on 11.12.2016]

Spahr, N., Hodkinson, D., Jolly, K., Williams, S., Howard, M., & Thacker, M., 2017. Distinguishing between nociceptive and neuropathic components in chronic low back pain using behavioural evaluation and sensory examination. *Musculoskeletal science & practice*, *27*, pp.40–48. Available from: doi:10.1016/j.msksp.2016.12.006 [Accessed on 15.06.2018]

Stankovic, A., Lazovic, M., Kocic, M., Dimitrijevic, L., Stankovic, I., Zlatavovic, D., 2012. Lumbar stabilization exercises in addition to strengthening and stretching exercises reduce pain and increase function in patients with chronic low back pain: Randomized clinical open-label study. [Online] *Turk. J. Phys. Med. Rehabil*. 58, pp.177-183. Available from: https://doi.10.42.4274/tftr.22438 [Accessed on 02.03.2015]

Stanton, T. Kawchuk, G., 2008. The effect of abdominal stabilization contractions on posteroanterior spinal stiffness. *Spine.*, 33(6), pp.694–701.

Staud R., 2013. The important role of CNS facilitation and inhibition for chronic pain. *International journal of clinical rheumatology*, *8*(6), pp.639–646.

Steffens, D., Hancock, M.J., Maher, C.G., Williams, C., Jensen, T.S., Latimer, J., 2014. Does magnetic resonance imaging predict future low back pain? A systematic review. *Eur. J. Pain*., 18(6), pp.755-765.

Stilwell, P., Harman, K., 2017. Contemporary biopsychosocial exercise prescription for chronic low back pain: questioning core stability programs and considering context. *J. Can. Chiropract. Ass.*, 61(1), pp.6-17.

Stokes, I.A., Gardner-Morse, M.G., Henry, S.M., 2011.Abdominal muscle activation increases lumbar spinal stability: analysis and contributions of different muscle groups. *Clin. Biomechan*., 26(8), pp.797-803.

Strong, J.A., Xie, W., Bataille, F.J., Zhang, J.M., 2013. Preclinical studies of low back pain. *Mol. Pain.*, 9(1), pp.17.

Sullivan, M.J., 2013. What is the clinical value of assessing pain-related psychosocial risk factors? *Pain Manage.*, 3(6), pp.413-416.

Sullivan, M.J., Adams, H., 2010. Psychosocial Treatment Techniques to Augment the Impact of Physiotherapy Interventions for Low Back Pain. *Physiother. Can*., 62(3), pp.180-189.

Sullivan, M.J.L., Adams, H., Horan, S., Maher, D., Boland, D., Gross, R., 2008. The role of perceived injustice in the experience of chronic pain and disability: Scale development and validation. *J Occup Rehabil.,* 18(3), pp.249-262.

Sullivan, M.J.L, Adams, H., Sullivan, M.E., 2004. Communicative dimensions of pain catastrophizing: social cueing effects on pain behaviour and coping. *Pain*., 107(3), pp.220-226.

Sullivan, M. J. L., Bishop, S. R., Pivik, J., 1995. The Pain Catastrophizing Scale: Development and validation. *Psych. Ass., 7*(4), pp.524-532.

Sullivan, M.J.L., Hyman, M.H., 2014. Return to work as a treatment objective for patients with chronic pain? *J. Pain Relief.*, 3, pp.1-3.

Sullivan, M.J., Lynch, M.E., Clark, A.J., 2005. Dimensions of catastrophic thinking associated with pain experience and disability in patients with neuropathic pain conditions. *Pain*., 113(3), pp.310-315.

Sullivan, M.J., Martel, M.O., Tripp, D.A., Savard, A., Crombez, G., 2006a. Catastrophic thinking and heightened perception of pain in others. *Pain*., 123(1-2), pp.37-44.

Sullivan, M.J., Martel, M.O., Tripp, D.A., Savard, A., Crombez, G., 2006b. The relation between catastrophizing and the communication of pain experience. *Pain.*, 122(3), pp.282-288.

Sullivan, M.J., Adams, H., Rhodenizer, T., Stanish, W.D., 2006c. A psychosocial risk factor--targeted intervention for the prevention of chronic pain and disability following whiplash injury. *Phys Ther*., 86(1), pp.8-18.

Sullivan, M.J.L., Rodgers, W.M., Kirsch, I., 2001b. Catastrophizing, depression and expectancies for pain and emotional distress. Pain. 91(1-2), pp.147-154.

Sullivan, M.J., Stanish, W., 2003. Psychological-based occupational rehabilitation: The Pain-Disability Prevention Program. *Clin. J. Pain.*, 24(19), pp.121-134.

Sullivan, M.J.L., Thibault, P., Andrikonyte, J., Butler, H., Catchlove, R., Lariviere, C., 2009. Psychological influences on repetition-induced summation of activity-related pain in patients with chronic low back pain. *Pain* ®, 141(1-2), pp.70-78.

Sullivan, M.J., Thorn, B., Haythornthwaite, J.A., Keefe, F., Martin, M., Bradley, L.A., Lefebvre, J.C., 2001a. Theoretical perspectives on the relation between catastrophizing and pain. *Clin. J. Pain*., 17(1), pp.52-64.

Sullivan, M.J.L., Ward, L.C., Trip, D., French, D.J., Adams, H., Stanish, W.D., 2005a. Secondary Prevention of Work Disability: Community-Based psychosocial Intervention for Musculoskeletal Disorders. *J. Occup. Rehabil*., 15(3), pp.377-392.

Suni, J., Rinne, M., Natri, A., Statistsian, M.P., Parkkari, J., Alaranta, H., 2006. Control of the lumbar neutral zone decreases low back pain and improves self-evaluated work ability: A 12-month randomized controlled study. *Spine*., 31(18), pp.611-620.

Suzuki, H., Aono, S., Inoue, S., Imajo, Y., Nishida, N., Nishida, N., Funaba, M., Harada, H., Mori, A., Matsumoto, M., Higuchi, F., Nakagawa, S., Tahara S., Ikeda, S., Izumi, H., Taguchi, T., Ushida, T., Sakai, T., 2020. Clinically significant changes in pain along the Pain Intensity Numerical Rating Scale in patients with chronic low back pain. PLOS ONE 15(3): e0229228. Available from: https://doi.org/10.1371/journal.pone.0229228 [Accessed 20.01.2021]

Swinkels, I.C.S., Wimmers, R.H., Groenewegen, R.P., van den Bosch, W.J.H., Dekker, J., van den Ende, C.H.M., 2005. What factors explain the number of physical therapy treatment sessions in patients referred with low back pain; a multilevel analysis. [Online] *BMC Health Services Res*., 5, 74. Available from: https://doi.org/10.1186/1472-6963-5-74 [Accessed 05.08.2017]

Tavel, M.E., 2014. Review. The placebo effect: the good, the bad, and the ugly. The American Journal of Medicine., 127(6), pp.484-488.

Taxonomy, I. A.S.P., 2014. *International Association for the Study of Pain*. [Online] Seattle: IASP Press. Available from: http://www.iasp-pain.org/Education/Content.aspx?ItemNumber=1698 [Accessed 16.06.2014]

Tonosu, J., Takeshita, K., Hara, N., Matsudaira, K., Kato, S., Masuda, K., Chikuda, H., 2012. The normative score and the cut-off value of the Oswestry Disability Index (ODI). *Eur. Spine.J.*, 21(8), pp.1596–1602.

Thompson, B. T., & Schoenfeld, D., 2007. Usual care as the control group in clinical trials of nonpharmacologic interventions. *Proceedings of the American Thoracic Society*, *4*(7), pp.577–582. Available from: https://doi.org/10.1513/pats.200706-072JK [Accessed 11.08.2017]

Thiese, M, S., Hegmann, K.T., Wood, E, M., Garg, A., Moore, J,S., Kapellusch, J., Foster, J., Ott, U., 2014. Prevalence of low back pain by anatomic location and intensity in an occupational population. *BMC Musculoskel. Disord*., 15(1), pp.283.

Thivel, D., Tremblay, A., Genin, P.M., Panahi, S., Rivière, D., Duclos, M., 2018. Physical Activity, Inactivity, and Sedentary Behaviors: Definitions and Implications in Occupational Health. *Frontiers in Public Health*., 6,. Available from: https://www.frontiersin.org/article/10.3389/fpubh.2018.00288 [Accessed 20.11.2020]

Torstensen, T.A., Ljunggren, A.E., Meen, H.D., Odland, E., Mowinckel, P., Geijerstam, S., 1998. Efficiency and costs of medical exercise therapy, conventional physiotherapy, and self-exercise in patients with chronic low back pain. A pragmatic, randomised, single-blinded controlled trial with 1 year follow up. *Spine*., 23(23), pp.2616-2624.

Tracey, I., 2016, Finding the hurt in pain. [Online] *Cerebrum: the Dana forum on brain science*. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5501013/ [Accessed 04.07.2017]

Tremblay, M. S., Aubert, S., Barnes, J. D., Saunders, T. J., Carson, V., Latimer-Cheung, A. E., Chastin, S., Altenburg, T. M., Chinapaw, M., & SBRN Terminology Consensus Project Participants (2017). Sedentary Behavior Research Network (SBRN) - Terminology Consensus Project process and outcome. *The international journal of behavioral nutrition and physical activity, 14*(1), 75.Available from: https://doi.org/10.1186/s12966-017-0525-8 [Accessed 25.12.2020]

Trompeter, K., Fett, D., Platen, P., 2017., Prevlence of Back Pain in Sports: A systematic Review of the literature. [Online] *Sports Med*., 47, pp.1183-1207. Available from: https//: doi:10.1007/s40279-016-0645-3. [Accessed 18.05.2018]

Tkachuk, G.A. and Harris, C.A., 2012. Psychometric properties of the Tampa Scale for Kinesiophobia-11 (TSK-11). *J. Pain.*, 13(10), pp.970-977.

Turk, D. C., Gatchel, R. J. Eds. 2002. *Psychological approaches to pain management: A practitioner's handbook*. 2nd ed. New York, US: Guilford Press

Turk, D.C., Okifuji, A., 2002. Psychological factors in chronic pain: evolution and revolution. *J. Consult Clin. Psychol.*, 70(3), pp.678-690.

Tudor-Locke, C.E., Ainsworth, B.E., Thompson, R.W., Matthews, C.E., 2002a. Comparison of pedometer and accelerometer measures of free-living physical activity. *Med. Sci. Sports Exerc*., 34(12), pp.2045-2051.

Tudor-Locke, C., Bassett, D.R., 2004. How many steps/day are enough. *Sports Med*., 34(1), pp1-8.

Tudor-Locke, C., Bassett, D.R., Shipe, M.F., McClain, J.J., 2011. Pedometry Methods for Assessing Free-Living Adults. *J. Phys. Act. Health*., 8(3), pp.445-453.

Tudor-Locke, C., Hatano, Y., Pangrazi, R.P., Kang, M., 2008. Revisiting "How Many Steps are enough?" Med. Sci. Sports Exerc., 40(7), pp.S537-S543.

Tudor-Locke, C., Hart, T. L., Washington, T. L., 2009. Correction: Expected values for pedometer-determined physical activity in older populations. *Int. J. Behav. Nutr. Phys. Act*, 6(1) pp.59.

Tudor-Locke, C.E., Myers, A.M., 2001a. Challenges and opportunities for measuring physical activity in sedentary adults. *Sports Med*., 31(12), pp.91-100.

Tudor-Locke, C.E., Myers, A.M., 2001b. Methodological considerations for researchers and practitioners using pedometers to measure (physical) ambulatory activity. *Res. Q Exerc. Sport*., 72(1), pp.1-12.

Tudor-Locke, C., Schuna, J. M., Jr., 2012. Steps to preventing type 2 diabetes: exercise, walk more, or sit less? [Online] Front. Endocrinol., 3(142), Available from: https://doi:10.3389/fendo.2012.00142 [Accessed 03.03.2019]

Tudor-Locke, C.E., Williams, J.E., Reis, J.P., Pluto, D., 2002b. Utility of pedometers for assessing physical activity. *Sports Med*., 32(12), pp.795-808.

Tuso, P., 2015. Strategies to Increase Physical Activity. *Permanente J.*, 19(4), pp.84–88. Available from: https//: doi:10.7812/TPP/14-242. [Accessed 11.03.2017]

Unsgaard-Tøndel, M., Fladmark, A.M., Salvesen, Ø., Vasseljen, O. 2010. Motor control exercises, sling exercises, and general exercises for patients with chronic low back pain: a randomized controlled trial with 1-year follow-up. *Phys Ther*., 90(10), pp.1426–1440.

Van Baar, M.E., Dekker, J., Bosveld, W., 1998. A survey of physical therapy goals and interventions for patients with back and knee pain. *Phys. Ther*., 78(1), pp.33-42.

Van den Hout, J.H.C., Vlaeyen, J.W.S., Heuts, P.H.T.G., Zijlema, J.H.L., Wijnen, J.A.G., 2003. Secondary Prevention of Work-Related Disability in Nonspecific Low Back Pain: Does problem Solving Therapy Help? A Randomized Clinical Trial. *Clin. J. Pain*., 19(2), pp.87-96.

Van Der Roer, N., Ostelo, R.W., Bekkering, G.E., Van Tulder, M.W., De Vet, H.C., 2006. Minimal clinically important change for pain intensity, functional status, and general health status in patients with nonspecific low back pain. *Spine.*, 31(5), pp.578-582.

Van der Heijden, G.J.M.G., Beurskens, A.JH.M., Dirx, M.J.M., Bouter, L.M., Lindeman, E., 1988. Efficacy of lumbar traction: A randomized controlled trial. Physiotherapy, 81., pp.29-35.

van Hecke, O., Kamerman, P.R., Attal, N., Baron, R., Bjornsdottir, G., Bennett, D.L.H, Bennett, M.I., Bouhassira, D., Diatchenko, L., Freeman, R., Freynhagen, R., Haanpää, M., Jensen, T.S., Raja, S.N., Rice, A.S.C., Seltzer, Z., Thorgeirsson, T.E., Yarnitsky, D., Smith, B.H., 2015. Neuropathic pain phenotyping by international consensus (NeuroPPIC) for genetic studies: a NeuPSIG systematic review, Delphi survey, and expert panel recommendations. *Pain.*, 156(11), pp.2337–2353.

Vancampfort, D., Stubbs, B., Koyanagi, A., 2017. Physical chronic conditions, multimorbidity and sedentary behavior amongst middle-aged and older adults in six low-and middle-income countries. *Int. J. Behav. Nutr. Phys Act.*, 14(1), pp.147.

van Middelkoop, M., Rubenstein, S.M., Kuijpers, T., Verhagen, A.P., Ostelo, R., Koes, B.W., van Tulder, M.W., 2011. A systematic review on the effectiveness of physical and rehabilitation interventions for chronic non-specific low back pain. *Eur. Spine J*., 20 (1), pp.19-39.

Van Slotten, T.T., Savelberg, H.H.C.M., Duimel-Peeters, I.G.P., Meijer, K., Henry, R.M.A., Stehouwer, C.D.A., Schaper, N.C., 2011. Peripheral neuropathy, decreased muscle strength and obesity are strongly associated with walking in persons with type 2 diabetes without manifest mobility limitations. *Diabetes Res. Clin. Pract.*, 91(1), pp.32-39.

Van Tulder, M., Becker, A., Bekkering, T., Breen, A., del Real, M,T, G., Hutchinson, A., Koes, B., Laerum, E., Malmivaara, A., 2006a. European guidelines for the management of acute nonspecific low back pain. *Eur. Spine J*., 15(Suppl. 2): S169-S191.

Van Tulder, M.W., Koes, B., Seitsalo, S., Malmivaara, A., 2006b. Outcome of invasive treatment modalities on back pain and sciatica: an evidence-based review. *Eur. Spine. J*., 15(1), pp.82-92.

Van Tulder, M.W., Koes, B.W., Malmivaara A.V., 2006c Outcome of non-invasive treatment modalities on back pain, an evidence based review. *Eur. Spine J*., 15(1), pp.64-81.

Van Tulder, M., Malmivaara, A., Esmail, R., Koes, B., 2000. Exercise therapy for low back pain: a systematic review within the framework of the Cochrane collaboration back review group. *Spine*., 25(21), pp.2784-2796.

Van Vuuren, B.J., van Heerden, H.J., Becker, P.J., Zinzen, E., Meeusen, R., 2006. Fear-avoidance beliefs and coping strategies in relation to lower back problems in a South African Steel Industry. *Eur. J. Pain*, 10(3), pp.233-239.

Van Weering, M.G., Vollenbroek-Hutten, M.M., Tonis, T.M., Hermens, H.J., van Weering, M.G.H., Vollenbroek-Hutten, M.M.R., 2009. Daily physical activities in chronic lower back pain patients assessed with accelerometery. *Eur J Pain.*, 13 (2009), pp. 649-654.

Vargas-Schaffer, G., 2010. Is the WHO analgesic ladder still valid: Twenty-four years of experience? *Can. Fam. Physician*, 56(6), pp.514-517.

Vela, L.I., Haladay, D.E., Denegar, C., 2011. Clinical assessment of low-back-pain treatment outcomes in athletes. *J Sport Rehabil.* 20(1), pp.74-88.

Verbunt, J.A., Westerterp, K.R., van der Heijden, G.J., Seelen, H.A., Vlaeyen, J.W., Knottnerus, J.A., 2001. Physical activity in daily life in patients with chronic low back pain. *Arch. Phys. Med. Rehabil*., 82(6), pp.726-730.

Verwoerd, A.J., Luijsterburg, P.A., Koes, B.W., el Barzouhi, A., Verhagen, A.P., 2015. Does Kinesiophobia Modify the Effects of Physical Therapy on Outcomes in Patients With Sciatica in Primary Care? Subgroup Analysis From a Randomized Controlled Trial. *Phys Ther*., 95(9), pp.1217-1223.

Vigotsky, A.D., Bruhns, R.P., 2015. The role of descending Modulation in Manual Therapy and its Analgesic Implications: A Narrative Review. [Online] *Pain Res. Treat*., Available from: https://doi:10.1155/2015/292805 [Accessed 03.02.2016]

Vlaeyen, J.W.S., de Jong, J.R., Onghena, P., Kerckoffs-Hanssen, M., Kole-Snijders, A.M.J., 2002. Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. *Pain Res. Manage.,* 7(3), pp.144-153.

Vlaeyen, J.W.S., Haazen, I.W.C.J., Schuerman, J.A., Kole-Snijders, A.M.J, van Eek, H., 1995. Behavioural rehabilitation of chronic low back pain: Comparison of an operant treatment, an operant-cognitive treatment and an operant-respondent treatment. *Br. J. Psychol.,* 34(1), pp.95-118.

Vogt, L., Pfeifer, K., Banzer, W., 2003. Neuromuscular control of walking with chronic low-back pain. *Man. Ther*., 8(1), pp.21-28.

von Hehn, C. A., Baron, R., Woolf, C. J., 2012. Deconstructing the neuropathic pain phenotype to reveal neural mechanisms. *Neuron.*, 73(4), pp.638–652. Available from: https//: doi:10.1016/j.neuron.2012.02.008 [Accessed 18.05.2016]

Voscopoulos, C., Lema, M., 2010. When does acute pain become chronic?. *Brit. J Anaesth.*, 105(suppl_1), pp.i69-i85.

Waddell, G. 2004. *The back pain revolution*. Edinburgh: Churchill Livingstone

Waddell, G., Newton, M., Henderson, I., Somerville, D., Main, C., 1993. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain*. 52(2)., pp.157-168.

Wall, P.D., Gutnick, M., 1974. Ongoing activity in peripheral nerves- physiology and pharmacology of impulses originating from a neuroma. *Exp. Neurol*., 43(3), pp.580-593.

Walker, B., 2000. The prevalence of Low Back Pain: A systematic review of the Literature from 1966 to 1998. *Clin. Spine Surg*., 13(3), pp.205-217.

Walker, B.F., Williamson, O.D., 2009. Mechanical or inflammatory low back pain. What are the potential signs and symptoms? *Man. Ther*., 14(3), pp.314-320.

Wand, B.M., O'Connell, N.E., 2008. Chronic non-specific low back pain- sub-groups or a single mechanism? *BMC Musculoskel. Disord*., 9(11), pp.11.

Wang, X.Q., Zheng, J.J., Yu, Z.W., Bi, X., Lou, S.J., Liu, J., Cai, B., Hua, Y.H., Wu, M., Wei, M.L., Shen, H.M., Chen, Y., Pan, Y.J., Xu, G.H., Chen, P.J., Eldabe S. 2012 A meta-analysis of core stability exercise versus general exercise for chronic low back pain.[Online] *PLoS One*., 7(12), e52082.

Warbuton, D.E., Bredin, S.S. 2017. Health benefits of physical activity: a systematic review of current systematic reviews. *Curr. Opin. Cardiol.*, 32(5), pp. 541-556.

Warburton, D.E., Bredin, S.S., Horita, L.T.L., Zbogar, D., Scott, J.M., Esch, B.T.A., Rhodes, R.E., 2007. The health benefits of interactive video game exercise. *Appl. Physiol. Nutr. Metab*., 32(4), pp.655-663.

Warburton, D.E., Nicol, C.W., Bredin, S.S., 2006. Health benefits of physical activity: the evidence. *CMAJ*,. 174(6), pp.801-809.

Wasfy, M. M., Baggish, A. L. 2016. Exercise Dose in Clinical Practice. *Circulation*, 133(23), pp.2297–2313

Watson, P.J., Booker, C.K., Moores, L., Main, C.J., 2004. Returning the chronically unemployed with low back pain to employment. *Eur J Pain*., 8(4), pp.359-369.

Weinstein, A.R., Sesso, H.D., 2006. Joint effects of physical activity and body weight on diabetes and cardiovascular disease. *Exerc. Sport Sci. Rev*., 34(1), pp.10–15.

Weishaupt, D., Zanetti, M., Hodler, J., Boos, N., 1998. MR imaging of the lumbar spine: prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. *Radiology*., 209(3), pp.661-666.

Weissman-Fogel, I., Sprecher, E., Pud, D., 2008. Effects of catastrophizing on pain perception and pain modulation. *Exp Brain Res*., 186(1). pp.79-85.

Westaway, M.S., Rheeder, P., Van Zyl, D.G., Seager, J.R., 2003. Interpersonal and organizational dimensions of patient satisfaction: the moderating effects of health status, *Int. J. Quality Health Care.*,15(4), pp.337–344.

Wiech, K., Ploner, M., Tracey, I., 2008. Review: Neurocognitive aspects of pain perception. *Trends Cog Sci*., 12(8), pp.306-313.

Wiech, K., Lin, C., Brodersen, K.H., Bingel, U., Ploner, M., Tracey, I., 2010. Anterior Insula Integrates Information about Salience into Perceptual Decisions about Pain. *J. Neurosci*., pp.16324-16331.

Whitlock, E.P., Orleans, C.T., Pender, N., Allan, J., 2002. Evaluating primary care behavioural counseling interventions: an evidence-based approach. *Am. J. Prev. Med.*, 22(4), pp.267-284.

Winter, E.M., Fowler, N., 2009. Exercise defined and quantified according to the Systeme International d'Unites [Online] *J. Sports Sci.*, 27(5), pp.447-460.

Woby, S.R., Roach, N.K., Urmston, M. and Watson, P.J., 2005. Psychometric properties of the TSK-11: a shortened version of the Tampa Scale for Kinesiophobia. *Pain.*, 117(1-2), pp.137-144.

Woby, S.R., Roach, N.K., Urmston, M. and Watson, P.J., 2008. Outcome following a physiotherapist-led intervention for chronic low back pain: the important role of cognitive processes. *Physiother.*, 94(2), pp.115-124.

Woodward, M., 2013. *Epidemiology: study design and data analysis*. 3rd ed. USA: CRC Press

Woolf, A., Pfelger, B., 2003. Burden of major musculoskeletal conditions. *Bulletin of the World Health Organization*., 81, pp.646-56. Available from: https//: doi: 10.1590/S0042-96862003000900007 [Accessed 13.06.2016]

Woolf, C.J., 2000. Pain. Neurobiology of disease, 7. pp.504-510.

Woolf, C.J., 2004. Dissecting out mechanisms responsible for peripheral neuropathic pain: implications for diagnosis and therapy. *Lif. Sci*., 74(21), pp.2605-2610.

Woolf, C.J., 2010. What is this thing called pain? *J. Clin. Invest*., 120(11), pp.3742-3744.

Woolf, C.J., 2011. Central sensitization: implications for the diagnosis and treatment of pain. *Pain.*, 152(3), pp.S2-15.

Woolf, C.J., Bennet, G.J., Doherty, M., Dubner, R., Kidd, B., Kolttzenburg, M., Lipton, R.,Loeser, J.D., Payne, R., Torebjork, E., 1998. Toward a mechanisms-based classification of pain? *Pain.*, 77(3), pp.227-229.

Woolf, C.J., Ma, Q., 2007. Nociceptors—noxious stimulus detectors. *Neuron*., 55(3), pp.353-364.

Woolf, C.J., Shorland, P., Reynolds, M., Ridings, J., Doubell, T., Coggeshall, R.E., 1995. Reorganization of central terminals of myelinated primary afferents in the rat dorsal horn following peripheral axotomy. *J. Comp. Neurol*., 360(1), pp.121-134.

Yamanouchi, K., Shinozaki, T., Chikada, K., Nishikawa, T., Ito, K., Shimizu, S., Ozawa, N., Suzuki, Y., Maeno, H., Kato, K., Oshida Y., 1995. Daily walking combined with diet therapy is a useful means for obese NIDDM patients not only to reduce body weight but also to improve insulin sensitivity. *Diabetes Care*., 18(6), pp.775-778.

Yang, H., Liu, H., Li, Z., Zhang, K., Wang, J., Wang, H., Zheng, Z., 2015. Low back pain associated with lumbar disc herniation: role of moderately degenerative disc and annulus fibrosis tears. *Int. J. Clin. Exp. Med.*, 8(2), pp.1634-1644.

Yoon, Y.W., Na, H.S., Chung, J.M., 1996. Contribution of injured and intact afferents to neuropathic pain in an experimental rat model. *Pain*., 64(1), pp.27-36.

Yoshiko, A., Tomita, A., Ando, R., Ogawa, M., Kondo, S., Saito, A., Tanaka, N. I., Koike, T., Oshida, Y., & Akima, H. (2018). Effects of 10-week walking and walking with home-based resistance training on muscle quality, muscle size, and physical functional tests in healthy older individuals. *European review of aging and physical activity : official journal of the European Group for Research into Elderly and Physical Activity*, *15*, 13. Available from : [https://doi.org/10.1186/s11556-018-0201-2] [Accessed 19.12.2020]

Yorkin, M., Spaccarotella, K., Martin-Biggers, J., Quick, V., Byrd-Bredbenner, C., 2013. Accuracy and consistency of weights provided by bathroom scales.[Online] *BMC Pub. Health*. Available from: https://doi.org/10.1186/1471-2458-13-1194 [Accessed 10.08.2017]

You, J.H., Kim, S.Y., Oh, D.W., Chon, S.C., 2014. The effect of a novel core stabilization technique on managing patients with chronic low back pain: A randomised, controlled, experimenter-blinded study. *Clin. Rehabil*., 28(5), pp.460-469.

Zusman, M., 2002. Forebrain-mediated sensitization of central pain pathways: 'non-specific' pain and a new image for MT. *Man Ther*.,7(2), pp.80-88.

Appendices

Appendix 1: University of Bath approval of ethics

Dear Richard,

Thank you for submitting the amendments requested by the REACH Committee. I can confirm these amendments were reviewed by Chair's action on 10th August and approved.

Please inform REACH about any substantial amendments made to the study if they have ethical implications.

Kind regards

Rachael Yates

Department Co-ordinator

Rachael McHugh, Department Co-ordinator
Department for Health
University of Bath
Building 1 West 4.112, Bath BA2 7AY, United Kingdom | Telephone: +44 (0)1225 383461 | Email: r.m.yates@bath.ac.uk

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Appendix 2: PACTR certificate

From: Elizabeth Pienaar [mailto:[redacted]]
Sent: 17 June 2016 11:30 AM
To: [redacted]
Subject: Trial registration on www.pactr.org - Approved
Importance: High

Dear Mr Feher

Re: A comparison of a pedometer-based walking program versus physiotherapy for patients suffering from nociceptive or neuropathic chronic, recurrent low back pain in Johannesburg

Please find attached confirmation of your registration with www.pactr.org; please ensure that you have read this letter and responded accordingly. Thank you for your application. We look forward to your registration of future trials.

Should you have any questions, please do not hesitate to contact me.

Regards

Elizabeth Pienaar
www.pactr.org Project Manager
Senior Scientist: Cochrane Centre
South African Medical Research Council
Tel: +[redacted] | Cell:
Francie van Zijl Drive, Parow Valley | Cape Town| Western Cape
www.samrc.ac.za

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Appendix 3: Pain and Activity Diary for W and PW treatment groups

Supported by:
UNIVERSITY OF BATH
SCHOOL FOR HEALTH

PAIN DIARY: DAY 1

SECTION A_1: PEDOMETER READING AND MEDICATION CHART

Please complete this pain diary before you go to bed every evening immediately after you have removed your pedometer. Write down the time in the evening when you record today's average pain. Please mark on the diagrams provided where your pains were today and the average amount of pain you felt in the area over the day.

PEDOMETER READING TODAY:

DATE	TIME (Pedometer removal)	TOTAL OF STEPS TODAY	DISTANCE WALKED TODAY

WALKING PROGRAMME PEDOMETER READING

TIME OF DAY WALKING PROGRAMME STARTED *(Please tick appropriate box)*	MORNING *04h00 – 11h59*	AFTERNOON *12h00 – 16h59*	EVENING 17h00 – 03h59
DURATION OF WALKING PROGRAM (MINS)			
NUMBER OF STEPS USED DURING WALKING PROGRAMME			
DISTANCE WALKED DURING WALKING PROGRAMME			

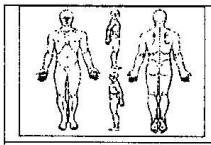
BACK PAIN DURING WALK *(Please tick appropriate box)*	YES	NO	INCREASED	DECREASED	UNCHANGED
LEG PAIN DURING WALK *(Please tick appropriate box)*	YES	NO	INCREASED	DECREASED	UNCHANGED

MEDICATION TAKEN TODAY *(if different from yesterday)*

TIME TAKEN	
NAME OF MEDICATION & DOSAGE TAKEN	

SECTION A_2: LOWER BACK PAIN AND LEG PAIN CHART AND VISUAL ANALOQUE SCALES

Please complete this pain diary before you go to bed every evening immediately after you have removed your pedometer. Write down the time in the evening when you record today's **average** pain. Please mark on the diagrams provided where your pains were today and the average amount of pain you felt in the area over the day.

	*	***Please mark in column***
	N	N = NONE
	I	I = INTERMITTENT
	C	C = CONSTANT
LOWER BACK PAIN		NO PAIN ——— VERY SEVERE PAIN
RIGHT LEG PAIN		NO PAIN ——— VERY SEVERE PAIN
LEFT LEG PAIN		NO PAIN ——— VERY SEVERE PAIN

Appendix 4: Pain and Activity Diary for P treatment group

Supported by:
UNIVERSITY OF BATH
SCHOOL FOR HEALTH

PAIN DIARY: DAY 1

SECTION A_1: PEDOMETER READING AND MEDICATION CHART

Please complete this pain diary before you go to bed every evening immediately after you have removed your pedometer. Write down the time in the evening when you record today's average pain. Please mark on the diagrams provided where your pains were today and the average amount of pain you felt in the area over the day.

PEDOMETER READING TODAY:

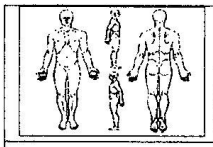
DATE	TIME (Pedometer removal)	TOTAL OF STEPS TODAY	DISTANCE WALKED TODAY

MEDICATION TAKEN TODAY ***(if different from yesterday)***

TIME TAKEN	
NAME OF MEDICATION & DOSAGE TAKEN	

SECTION A_2: LOWER BACK PAIN AND LEG PAIN CHART AND VISUAL ANALOQUE SCALES

Please complete this pain diary before you go to bed every evening immediately after you have removed your pedometer. Write down the time in the evening when you record today's **average** pain. Please mark on the diagrams provided where your pains were today and the average amount of pain you felt in the area over the day.

	*	**** Please mark in column***
	N	N = NONE
	I	I = INTERMITTENT
	C	C = CONSTANT
LOWER BACK PAIN		NO PAIN ——— VERY SEVERE PAIN
RIGHT LEG PAIN		NO PAIN ——— VERY SEVERE PAIN
LEFT LEG PAIN		NO PAIN ——— VERY SEVERE PAIN

Appendix 5: Health history and demographic questionnaire

HEALTH HISTORY and DEMOGRAPHIC QUESTIONNAIRE

Please answer the following health questions regarding your physical health:

If you wish to add any details, please do so in the additional information column. Please ask the physiotherapist to explain anything below if you have any questions.

	YES	NO	ADDITIONAL INFORMATION
Age > 70 years			
Minor trauma if age > 50 years			
Significant trauma			
History of recent infection			
Penetrating wound near the spine			
Night pain			
Pain at rest			
Unexplained weight loss			
History of cancer			
Progressive or disabling neurological symptoms/ signs (numbness between your legs, bilateral sciatica or leg weakness, urinary retention, faecal incontinence			
History of osteoporosis			
Intravenous drug abuse			
Long term corticosteroid use			
Non responsive to conservative treatment after six weeks			
Immunocompromised			
Current Pregnancy			
Current Malignancy			
Rheumatic disease			
Diagnosed with Fibromyalgia			
Known Psychological disorders			
Current spinal fracture			
Medically unfit to participate in exercise program			
Are you a smoker?			
Are you able to walk a minimum of 20 minutes			
Level of education (no matric, matric, diploma, degree, post graduate)			
Age (years), Gender, Ethnicity			
Employed (yes/no)			
Smoker (yes/no)			
Height (cm)/ Weight(kg)			
Married (yes/no); n.o.children			

Appendix 6: painDETECT questionnaire

painDETECT

PAIN QUESTIONNAIRE

Date: _____ Patient: Last name: _____ First name: _____

How would you assess your pain **now**, at this moment?

0 1 2 3 4 5 6 7 8 9 10

none max.

How strong was the **strongest** pain during the past 4 weeks?

0 1 2 3 4 5 6 7 8 9 10

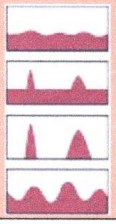
none max.

How strong was the pain during the past 4 weeks **on average**?

0 1 2 3 4 5 6 7 8 9 10

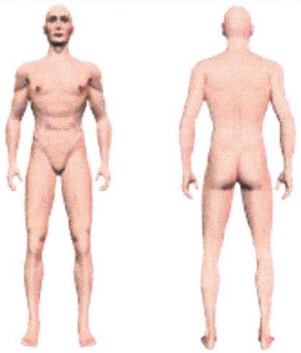
none max.

Mark the picture that best describes the course of your pain:



- **Persistent pain with slight fluctuations** ☐
- **Persistent pain with pain attacks** ☐
- **Pain attacks without pain between them** ☐
- **Pain attacks with pain between them** ☐

Please mark your **main area of pain**



Does your pain radiate to other regions of your body? yes ☐ no ☐

If yes, please draw the direction in which the pain radiates.

Do you suffer from a burning sensation (e.g., stinging nettles) in the marked areas?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Do you have a tingling or prickling sensation in the area of your pain (like crawling ants or electrical tingling)?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Is light touching (clothing, a blanket) in this area painful?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Do you have sudden pain attacks in the area of your pain, like electric shocks?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Is cold or heat (bath water) in this area occasionally painful?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Do you suffer from a sensation of numbness in the areas that you marked?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

Does slight pressure in this area, e.g., with a finger, trigger pain?

never ☐ hardly noticed ☐ slightly ☐ moderately ☐ strongly ☐ very strongly ☐

(To be filled out by the physician)

never	hardly noticed	slightly	moderately	strongly	very strongly
☐ x 0 = 0	☐ x 1 = ☐	☐ x 2 = ☐☐	☐ x 3 = ☐☐	☐ x 4 = ☐☐	☐ x 5 = ☐☐

Total score ☐☐ **out of 35**

Development/Reference: R. Freynhagen, R. Baron, U. Gockel, T.R. Tölle / Curr Med Res Opin, Vol.22, No. 10 (2006)

painDETECT questionnaire, ©2005 Pfizer Pharma GmbH, used with permission.

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painDETECT

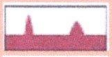
SCORING OF PAIN QUESTIONNAIRE

Date: _____ Patient: Last name: _____ First name: _____

Please transfer the total score from the pain questionnaire:

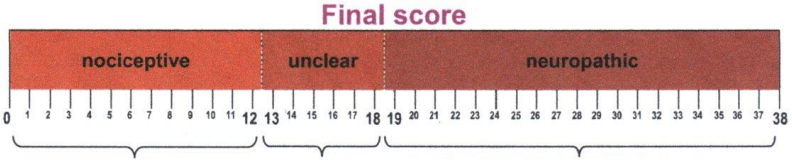
Total score ☐☐

Please add up the following numbers, depending on the marked pain behavior pattern and the pain radiation. Then total up the final score:

	Persistent pain with slight fluctuations	0
	Persistent pain with pain attacks	- 1 if marked, or
	Pain attacks without pain between them	+ 1 if marked, or
	Pain attacks with pain between them	+ 1 if marked
	Radiating pains?	+ 2 if yes
	Final score	☐☐

Screening Result

Final score



A neuropathic pain component is unlikely (< 15%)	Result is ambiguous, however a neuropathic pain component can be present	A neuropathic pain component is likely (> 90%)

This sheet does not replace medical diagnostics.
It is used for screening the presence of a neuropathic pain component.

Development/Reference: R. Freynhagen, R. Baron, U. Gockel, T.R. Tölle / Curr Med Res Opin, Vol.22, No. 10 (2006)

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Appendix 7: Patient Centred Outcome Questionnaire

APPENDIX A
PATIENT-CENTERED OUTCOMES QUESTIONNAIRE (PCOQ)

MANY PEOPLE EXPERIENCE PAIN, FATIGUE (I.E., FEELING TIRED), EMOTIONAL DISTRESS (E.G., WORRIES, FEELING SAD), AND INTERFERENCE WITH DAILY ACTIVITIES (E.G., NOT BEING ABLE TO WORK OR DO HOUSEHOLD CHORES) AS A RESULT OF THEIR MEDICAL CONDITION. WE WOULD LIKE TO UNDERSTAND HOW YOU HAVE BEEN IMPACTED IN EACH OF THESE AREAS. WE WOULD ALSO LIKE TO LEARN MORE ABOUT WHAT YOU WANT YOUR TREATMENT TO DO FOR YOU.

FIRST, WE WOULD LIKE TO KNOW YOUR **USUAL** LEVELS OF PAIN, FATIGUE, EMOTIONAL DISTRESS, AND INTERFERENCE.

On a scale of ***0 (none)*** **to** ***100 (worst imaginable)***, please indicate your usual level (during the past week) of …

- pain _____
- fatigue (or tiredness) _____
- emotional distress _____
- interference with daily activities _____

NOW, WE WOULD LIKE TO LEARN ABOUT YOUR **DESIRED** LEVELS OF PAIN, FATIGUE, EMOTIONAL DISTRESS, AND INTERFERENCE. IN OTHER WORDS, WE WOULD LIKE TO UNDERSTAND WHAT YOUR IDEAL TREATMENT OUTCOME WOULD BE.

On a scale of ***0 (none)*** **to** ***100 (worst imaginable)***, please indicate your desired level of …

- pain _____
- fatigue (or tiredness) _____
- emotional distress _____
- interference with daily activities _____

PATIENTS UNDERSTANDABLY WANT THEIR TREATMENT TO RESULT IN DESIRED OR IDEAL OUTCOMES LIKE YOU INDICATED ABOVE. UNFORTUNATELY, AVAILABLE TREATMENTS DO NOT ALWAYS PRODUCE DESIRED OUTCOMES. THUS, IT IS IMPORTANT FOR US TO UNDERSTAND WHAT TREATMENT OUTCOMES YOU WOULD CONSIDER **SUCCESSFUL**.

On a scale of ***0 (none)*** **to** ***100 (worst imaginable)***, please indicate the level each of these areas would have to be at for you to consider treatment successful.

- pain _____
- fatigue (or tiredness) _____
- emotional distress _____
- interference with daily activities _____

NOW, WE WOULD LIKE TO KNOW WHAT YOU **EXPECT** YOUR TREATMENT TO DO FOR YOU.

On a scale of ***0 (none)*** **to** ***100 (worst imaginable)***, please indicate the levels you expect following treatment.

- pain _____
- fatigue (or tiredness) _____
- emotional distress _____
- interference with daily activities _____

FINALLY, WE WOULD LIKE TO UNDERSTAND HOW **IMPORTANT** IT IS FOR YOU TO SEE IMPROVEMENT IN YOUR PAIN, FATIGUE, EMOTIONAL DISTRESS, AND INTERFERENCE FOLLOWING TREATMENT.

On a scale of ***0 (not at all important)*** **to** ***100 (most important)***, please indicate how important it is for you to see improvement in your…

- pain _____
- fatigue (or tiredness) _____
- emotional distress _____
- interference with daily activities _____

Appendix 8: Lumbar assessment

UNIVERSITY OF BATH
SCHOOL FOR HEALTH

Professional Doctorate in Health

Principal Researchers Name: **RICHARD FEHER**
BSc (Physiotherapy) WITS
MPhil (Sports Physiotherapy) UCT
E-mail: [redacted]
Tel: [redacted]

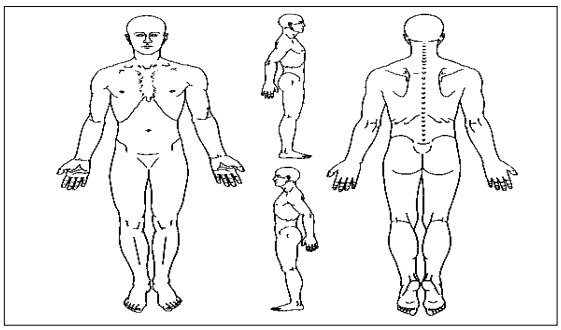
Title of the Project: A comparison of a pedometer-based walking program versus physiotherapy for patients suffering from primarily nociceptive or neuropathic chronic low back pain in Johannesburg.

Complex evaluation involving a subjective and objective lower back pain examination.

Lumbar Evaluation Form

Patient Name		Eval Date	
Physician		DOB	
Therapist		Next MD visit	
Gender (M/F)		Ethnicity	

LUMBAR OBSERVATION



From Behind:	L Side: R Side:

ACTIVE MOVEMENTS

Flexion:	Extension:
R Lateral flexion: L Lateral flexion:	R Rotation: L Rotation:
Quadrant/Combined movement:	R Single leg extension : L Single leg extension:

PASSIVE MOVEMENTS (ALL ACTIVE MOVEMENTS WITH OVER PRESSURE)

Flexion:	Extension:
R Lateral flexion: L Lateral flexion:	R Rotation: L Rotation:
Quadrant/Combined movement:	R Single leg extension : L Single leg extension:

MUSCLE LENGTH

R Psoas: L Psoas:
R Hamstring: L Hamstring:
R Gluteal: L Gluteal:

HIP QUADRANT

R Hip:
L Hip:

Pt History of Pain/Symptoms

1. Onset of Sx's → ❑ **Gradual** ❑ **Sudden** If sudden, was there a specific event/injury?

2. Pain Level lower back → **Current** pain ____/10 **Worst** pain _____/10 **Best** pain _____/10

Pain Level R Leg → **Current** pain ____/10 **Worst** pain _____/10 **Best** pain _____/10

Pain Level L Leg → **Current** pain ____/10 **Worst** pain _____/10 **Best** pain _____/10

3. Pain Type lower back → ❑ **Aching** ❑ **Dull** ❑**Tingling** ❑ **Stabbing** ❑ **Burning** ❑ **Nauseating** ❑**Other:**

Pain Type R Leg → ❑ **Aching** ❑ **Dull** ❑**Tingling** ❑ **Stabbing** ❑ **Burning** ❑ **Nauseating** ❑**Other:**

Pain Type L Leg → ❑ **Aching** ❑ **Dull** ❑**Tingling** ❑ **Stabbing** ❑ **Burning** ❑ **Nauseating** ❑**Other:**

4. What **relieves** pain/Sxs?

Pain lower back :

Pain R Leg :

Pain L Leg :

(positions, movements meds, modalities)

5. What makes pain/Sxs **worse**?

Pain lower back :

Pain R Leg :

Pain L Leg :

(positions, movements, activities)

6. LBP Pain/Sx's. Frequency:

❑ **Intermittent** ❑ **Constant**

Leg Pain/Sx's. Frequency:

❑ **Intermittent** ❑ **Constant**

7. LBP Duration of Pain/Sx's:

❑ **< 16 days** ❑ **> 16 days**

8. Leg Duration of Pain/Sx's:

❑ **< 16 days** ❑ **> 16 days**

9. LBP Pain/Sx's worse:

10. ❑ **In Morning** ❑ **At Night**

11. Leg Pain/Sx's worse:

❑ **In Morning** ❑ **At Night**

12. Symptoms below the knee?

YES NO

IF YES → PERFORM LOWER QUARTER SCREEN

IF NO → PERFORM SI/PELVIC ASSESSMENT

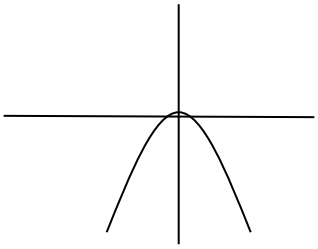
LOWER QUARTER SCREEN

	Muscle Testing		**Sensory Testing** *(Intact / Diminished / Absent)*		**Special Tests**	**Right**	**Left**
	Right	**Left**	**Right**	**Left**			
L1/L2 (Hip flex)					**Patellar DTR** (L3-4) *(Hypo 1+, Normal 2+, Hyper 3+, Clonus 4+)*		
L3/L4 (Quads)					**Achilles DTR** (S1-2) *(Hypo 1+, Normal 2+, Hyper 3+, Clonus 4+)*		
L4/L5 (Ant Tib)					**Babinski** (+ or -)		
L5 (EHL)					**Clonus** (If +, # of beats)		
L5/S1 (Evertors)					**SLR** (+ or -) for recreation of **"their"** pain/sx's		
S1/S2 (PF'ers)							

SI/PELVIC ASSESSMENT

Initial SI Test
1. PSIS Levels in Sitting: + -
2. Standing Forward Flexion: + -
3. Supine to Sit: + -
4. Prone Knee Flexion: + -
Total positive: **/4**

ROM

	Range *(Full or % Limited)*	**Limited By** *(Pain, mm tightness, etc)*	**Deviations?**	
Flexion				
Extension				
R SB'ing				
L SB'ing				
R Rotation				
L Rotation				

JOINT MOBILITY PALPATION

Level	**Central PA** *(Hypo, N, Hyper)*	**L Unilateral/ Apophoseal** *(Hypo, N, Hyper)*	**R Unilateral/ Apophoseal** *(Hypo, N, Hyper)*	**Pain w/ assessment?**	**Does it recreate "their" pain?**
T12					
L1					
L2					
L3					
L4					
L5					

JOINT MOBILITY PALPATION

Level	**LTransverse Process** *(Hypo, N, Hyper)*	**RTransverse Process** *(Hypo, N, Hyper)*	**Pain w/ assessment?**	**Does it recreate "their" pain?**
T12				
L1				
L2				
L3				
L4				
L5				

PALPATION

PARASPINAL MUSCLES	**QUADRATUS LUMBORUM MUSCLES**	**GLUTEAL MUSCLES**	**Pain w/ assessment?**	**Does it recreate "their" pain?**

SPECIAL TESTS:

- STRAIGHT LEG RAISE TEST/ SLUMP:
- PRONE KNEE BEND/FEMORAL SLUMP:
- SACRO-ILLIAC JOINT TEST:
- NEUROLOGICAL EXAMINATION:
- X-RAY
- BONE SCAN
- CT SCAN
- MRI SCAN

Appendix 9: Numerical Rating Scale for pain at baseline

NUMERICAL RATING SCALE (NRS) FOR PAIN INENSITY ZERO WEEKS – BASE LINE

FIRST, WE WOULD LIKE TO KNOW YOUR **USUAL** LEVEL OF PAIN

On a scale of ***0 (none) to 100 (worst imaginable)***, please indicate your usual level (during the past week) of:

- Pain _____

Appendix 10: Numerical Rating Scale for pain at six-weeks

NUMERICAL RATING SCALE (NRS) FOR PAIN INENSITY SIX-WEEKS

FINALLY, WE WOULD LIKE TO KNOW YOUR **USUAL** LEVEL OF PAIN **FOLLOWING TREATMENT**. IN OTHER WORDS, WHAT ARE YOUR CURRENT LEVELS OF PAIN?

On a scale of ***0 (none) to 100 (worst imaginable)***, please indicate your usual level (during the past week) of:

- Pain _____

Appendix 11: Numerical Rating Scale for pain at 12-weeks

NUMERICAL RATING SCALE (NRS) FOR PAIN INENSITY TWELVE-WEEKS

FINALLY, WE WOULD LIKE TO KNOW YOUR **USUAL** LEVEL OF PAIN **FOLLOWING TREATMENT**. IN OTHER WORDS, WHAT ARE YOUR CURRENT LEVELS OF PAIN?

On a scale of ***0 (none) to 100 (worst imaginable)***, please indicate your usual level (during the past week) of:

- Pain _____

Appendix 12: Oswestry Disability Index questionnaire

Oswestry Low Back Pain Disability Questionnaire

Sources: Fairbank JCT & Pynsent, PB (2000) The Oswestry Disability Index. *Spine*, 25(22):2940-2953.

Davidson M & Keating J (2001) A comparison of five low back disability questionnaires: reliability and responsiveness. *Physical Therapy* 2002;82:8-24.

The Oswestry Disability Index (also known as the Oswestry Low Back Pain Disability Questionnaire) is an extremely important tool that researchers and disability evaluators use to measure a patient's permanent functional disability. The test is considered the 'gold standard' of low back functional outcome tools [1].

Scoring instructions

For each section the total possible score is 5: if the first statement is marked the section score = 0; if the last statement is marked, it = 5. If all 10 sections are completed the score is calculated as follows:

Example: 16 (total scored)

50 (total possible score) x 100 = 32%

If one section is missed or not applicable the score is calculated:

16 (total scored)

45 (total possible score) x 100 = 35.5%

Minimum detectable change (90% confidence): 10% points (change of less than this may be attributable to error in the measurement)

Interpretation of scores

0% to 20%: minimal disability:	The patient can cope with most living activities. Usually no treatment is indicated apart from advice on lifting sitting and exercise.
21%-40%: moderate disability:	The patient experiences more pain and difficulty with sitting, lifting and standing. Travel and social life are more difficult and they may be disabled from work. Personal care, sexual activity and sleeping are not grossly affected and the patient can usually be managed by conservative means.
41%-60%: severe disability:	Pain remains the main problem in this group but activities of daily living are affected. These patients require a detailed investigation.
61%-80%: crippled:	Back pain impinges on all aspects of the patient's life. Positive intervention is required.
81%-100%:	These patients are either bed-bound or exaggerating their symptoms.

Oswestry Low Back Pain Disability Questionnaire

Instructions

This questionnaire has been designed to give us information as to how your back or leg pain is affecting your ability to manage in everyday life. Please answer by checking ONE box in each section for the statement which best applies to you. We realise you may consider that two or more statements in any one section apply but please just shade out the spot that indicates the statement which most clearly describes your problem.

Section 1 – Pain Intensity

- ☐ I have no pain at the moment
- ☐ The pain is very mild at the moment
- ☐ The pain is moderate at the moment
- ☐ The pain is fairly severe at the moment
- ☐ The pain is very severe at the moment
- ☐ The pain is the worst imaginable at the moment

Section 2 – Personal care (washing, dressing etc)

- ☐ I can look after myself normally without causing extra pain
- ☐ I can look after myself normally but it causes extra pain
- ☐ It is painful to look after myself and I am slow and careful
- ☐ I need some help but manage most of my personal care
- ☐ I need help every day in most aspects of self-care
- ☐ I do not get dressed, I wash with difficulty and stay in bed

Section 3 – Lifting

- ☐ I can lift heavy weights without extra pain
- ☐ I can lift heavy weights but it gives extra pain
- ☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently placed eg. on a table
- ☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- ☐ I can lift very light weights
- ☐ I cannot lift or carry anything at all

Section 4 – Walking*

- ☐ Pain does not prevent me walking any distance
- ☐ Pain prevents me from walking more than 1 mile
- ☐ Pain prevents me from walking more than 1/2 mile
- ☐ Pain prevents me from walking more than 100 yards
- ☐ I can only walk using a stick or crutches
- ☐ I am in bed most of the time

Section 5 – Sitting

- ☐ I can sit in any chair as long as I like
- ☐ I can only sit in my favourite chair as long as I like
- ☐ Pain prevents me sitting more than one hour
- ☐ Pain prevents me from sitting more than 30 minutes
- ☐ Pain prevents me from sitting more than 10 minutes
- ☐ Pain prevents me from sitting at all

Section 6 – Standing

- ☐ I can stand as long as I want without extra pain
- ☐ I can stand as long as I want but it gives me extra pain
- ☐ Pain prevents me from standing for more than 1 hour
- ☐ Pain prevents me from standing for more than 30 minutes
- ☐ Pain prevents me from standing for more than 10 minutes
- ☐ Pain prevents me from standing at all

Section 7 – Sleeping

- ☐ My sleep is never disturbed by pain
- ☐ My sleep is occasionally disturbed by pain
- ☐ Because of pain I have less than 6 hours sleep
- ☐ Because of pain I have less than 4 hours sleep
- ☐ Because of pain I have less than 2 hours sleep
- ☐ Pain prevents me from sleeping at all

Section 8 – Sex life (if applicable)

- ☐ My sex life is normal and causes no extra pain
- ☐ My sex life is normal but causes some extra pain
- ☐ My sex life is nearly normal but is very painful
- ☐ My sex life is severely restricted by pain
- ☐ My sex life is nearly absent because of pain
- ☐ Pain prevents any sex life at all

Section 9 – Social life

- ☐ My social life is normal and gives me no extra pain
- ☐ My social life is normal but increases the degree of pain
- ☐ Pain has no significant effect on my social life apart from limiting my more energetic interests eg, sport
- ☐ Pain has restricted my social life and I do not go out as often
- ☐ Pain has restricted my social life to my home
- ☐ I have no social life because of pain

Section 10 – Travelling

- ☐ I can travel anywhere without pain
- ☐ I can travel anywhere but it gives me extra pain
- ☐ Pain is bad but I manage journeys over two hours
- ☐ Pain restricts me to journeys of less than one hour
- ☐ Pain restricts me to short necessary journeys under 30 minutes
- ☐ Pain prevents me from travelling except to receive treatment

References

1. Fairbank JC, Pynsent PB. The Oswestry Disability Index. Spine 2000 Nov 15;25(22):2940-52; discussion 52.

Appendix 13: Tampa Scale for Kinesiophobia

Tampa Scale for Kinesiophobia
(Miller , Kori and Todd 1991)

1 = strongly disagree
2 = disagree
3 = agree
4 = strongly agree

1. I'm afraid that I might injury myself if I exercise	1	2	3	4
2. If I were to try to overcome it, my pain would increase	1	2	3	4
3. My body is telling me I have something dangerously wrong	1	2	3	4
4. My pain would probably be relieved if I were to exercise	1	2	3	4
5. People aren't taking my medical condition seriously enough	1	2	3	4
6. My accident has put my body at risk for the rest of my life	1	2	3	4
7. Pain always means I have injured my body	1	2	3	4
8. Just because something aggravates my pain does not mean it is dangerous	1	2	3	4
9. I am afraid that I might injure myself accidentally	1	2	3	4
10. Simply being careful that I do not make any unnecessary movements is the safest thing I can do to prevent my pain from worsening	1	2	3	4
11. I wouldn't have this much pain if there weren't something potentially dangerous going on in my body	1	2	3	4
12. Although my condition is painful, I would be better off if I were physically active	1	2	3	4
13. Pain lets me know when to stop exercising so that I don't injure myself	1	2	3	4
14. It's really not safe for a person with a condition like mine to be physically active	1	2	3	4
15. I can't do all the things normal people do because it's too easy for me to get injured	1	2	3	4
16. Even though something is causing me a lot of pain, I don't think it's actually dangerous	1	2	3	4
17. No one should have to exercise when he/she is in pain	1	2	3	4

Reprinted from:
Pain, Fear of movement/(re) injury in chronic low back pain and its relation to behavioral performance, 62, Vlaeyen, J., Kole-Snijders A., Boeren R., van Eek H., 371.
Copyright (1995) with permission from International Association for the Study of Pain.

Scoring Information
Tampa Scale for Kinesiophobia
(Miller et al 1991)

A total score is calculated after inversion of the individual scores of items 4, 8, 12 and 16.

Reprinted from:
Pain, Fear of movement/(re) injury in chronic low back pain and its relation to behavioral performance, 62, Vlaeyen, J., Kole-Snijders A., Boeren R., van Eek H., 371.
Copyright (1995) with permission from International Association for the Study of Pain.

Appendix 14: Pain Catastrophizing Scale

Copyright © 1995
Michael JL Sullivan

PCS-EN

Client No.: _____ Age: _____ Sex: M(__) F(__) Date: _____

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

0 – not at all **1** – to a slight degree **2** – to a moderate degree **3** – to a great degree **4** – all the time

When I'm in pain ...

1. ☐ I worry all the time about whether the pain will end.
2. ☐ I feel I can't go on.
3. ☐ It's terrible and I think it's never going to get any better.
4. ☐ It's awful and I feel that it overwhelms me.
5. ☐ I feel I can't stand it anymore.
6. ☐ I become afraid that the pain will get worse.
7. ☐ I keep thinking of other painful events.
8. ☐ I anxiously want the pain to go away.
9. ☐ I can't seem to keep it out of my mind.
10. ☐ I keep thinking about how much it hurts.
11. ☐ I keep thinking about how badly I want the pain to stop.
12. ☐ There's nothing I can do to reduce the intensity of the pain.
13. ☐ I wonder whether something serious may happen.

...Total

Updated 11/11

Appendix 15: Participant information sheet

Professional Doctorate in Health

Principal Researchers Name: **RICHARD FEHER**

BSc (Physiotherapy) WITS

MPhil (Sports Physiotherapy) UCT

E-mail: r[redacted]

Tel: [redacted]

Title of the Project: A comparison of a pedometer-based walking program versus physiotherapy for patients suffering from nociceptive or neuropathic chronic, recurrent low back pain in Johannesburg.

Letter to study participant/ patient.

Dear Participant

We are investigating three different treatments for patients with chronic and recurrent lower back pain. The study is being run by Richard Feher (principal investigator) studying his Doctorate in Health at the University of Bath, Department of Health (United Kingdom). Patients who attend the Medicross Randburg, Dr Guy Teuwen Private Practice Neurosurgical Rooms or Towers West Medical Centre have been invited to participate.

We would like to investigate which specific pain types respond best to preferred treatments for your pain. After you have given us your consent, using the painDETECT questionnaire, you will receive a specific diagnosis of your chronic recurrent lower back pain. You will then be randomly allocated to one of three different treatment programmes.

We will then conduct a clinical examination which will involve the following:

1. You will complete a form telling us about your general health.
2. You will be asked a series of questions about your pain and function, and its effect on your life.
3. The physiotherapist will examine your back using their hands as well as asking you to perform some activities using your lower back and your legs.

Following the above steps, you will be treated by a qualified physiotherapist. The treatment program will last for 12-weeks. During this time you will visit the physiotherapist between 3 and 9 times depending on your individual needs. You will receive, and be shown how to use a pain and activity diary and pedometer. It is important that you wear the pedometer from the moment you put your clothes on in the begging of the day until you go to bed at night, in order for your correct step count to be measured. The pedometer will record step counts, distance and time. The diary must be completed every day before you go to sleep in the evening. Please record your average daily pain and the areas where you felt the pain in the spaces provided on each diary page. There will also be spaces to fill in your step counts and distance you walked in the day. Only fill in changes in medication taken if it differs from what you recorded on your very first entry. The diary should be filled out accurately and honestly over the next 12-weeks, with the completed diary handed in to your physiotherapist on your last physiotherapy appointment for the study to be successful. For the purposes of this study, only completed diaries will be accepted. If you do not hand in your pain diary on the last physiotherapy appointment, you will be contacted either telephonically or by e mail within 2 weeks after the predicted date of your last appointment. If you still cannot be reached, then you will be excluded from the study.

Direct benefits to you may include decreased pain during and after the study. Additional benefit may be shown in improved function with activities of daily living. Indirect benefits suggested by the study may be matching specific physiotherapy treatments to different types of pain. Since the study involves the participation of people who are suffering with chronic and/or recurrent lower back pain, the risk therefore involves increasing your levels of pain with participation in various physiotherapy treatments. The treatments aim to decrease your pain and improve your function over the 12-week period. If pain starts to increase, let your attending physiotherapist know. It is important that we try not to let your pain increase when participating in the study. Since this study involves participants suffering chronic pain, it is important to note that compensation and free medical treatment is not generally owed to research participants who suffer expected or foreseen adverse reactions to investigational therapeutic, diagnostic or preventative treatment when such reactions are not different in kind from those known to be associated with established interventions in standard medical practice. If however, impairment, disability or handicap as consequence of one’s participation in the study results, you will be entitled to free medical treatment and compensation.

You may withdraw from the study at any time without prejudice or penalty, but are kindly asked to contact the researcher if a decision is taken to do so. If you wish to withdraw from the study, please fill out section B in your pain diary. It will be more beneficial if more patients complete the study. Your data will only be accessed by the primary researcher as well as the treating physiotherapist allocated to you. Information regarding your treatments will be kept anonymous and safely stored in a safe on the premises of the practice where the research and treatments are to take place. The data will be kept in a secure location after the completion of the study by the principal researcher. If you have any queries before participation or during the study, do not hesitate to contact me on the numbers provided below.

Researchers Contact information

Investigator Name	Contact Number	Email address
Richard Feher (Principal)	0117913454	richphysio@vodamail.co.za
Ross Cartwright	0117913454	physiofeher@telkomsa.net
Heather Brooker	0117913454	physiofeher@telkomsa.net
Joshua Sandler	0117913454	physiofeher@telkomsa.net
Jessica Rabbitte	0117913454	physiofeher@telkomsa.net
Sandra Laubscher	0117913454	physiofeher@telkomsa.net
Nikki Coghill (Supervisor)		n.coghill@bath.ac.uk
Antonia Wadley(Practice-based Supervisor)		antonia.wadley@wits.ac.za

This study has been reviewed and received ethics clearance through the Research Ethics Approval Committee for Health.

Kind regards

Richard Feher

Appendix 16: Participant consent form

Professional Doctorate in Health

Principal Researchers Name: RICHARD FEHER

BSc (Physiotherapy) WITS

MPhil (Sports Physiotherapy) UCT

E-mail: r[redacted]

Tel: [redacted]

Title of the Project: A comparison of a pedometer-based walking program versus physiotherapy for patients suffering from nociceptive or neuropathic chronic, recurrent low back pain in Johannesburg.

Informed consent form

Dear Participant

The University of Bath and doctoral student, Richard Feher, will be conducting a study to investigate the following:

- To assess whether an allocated program will decrease lower back and/or leg pain in either or both of the different pain (pheno)type groups.
- To assess if an allocated program will increase function in either or both of the different pain (pheno) type groups.
- To assess which treatment group and/or pain phenotype best matches treatment expectation to outcome.

The study will help us understand how much walking is necessary regardless of physiotherapy treatment, to help people like yourself with pain and movement.

Researchers Contact information

Investigator Name	Contact Number	Email address
Richard Feher (Principal)	0117913454	richphysio@vodamail.co.za
Ross Cartwright	0117913454	physiofeher@telkomsa.net
Heather Brooker	0117913454	physiofeher@telkomsa.net
Sandra Laubscher	0117913454	physiofeher@telkomsa.net
Joshua Sandler	0117913454	physiofeher@telkomsa.net
Jessica Rabbitte	0117913454	physiofeher@telkomsa.net
Nikki Coghill (Supervisor)		n.coghill@bath.ac.uk
Antonia Wadley (Local Supervisor)		antonia.wadley@wits.ac.za

Please answer all the following questions by ticking the appropriate block. Once all the questions have been answered please print your name and sign the form in the space provided.

Consent Form

Have you read the study information sheet?	**Yes**	**No**
Have you had an opportunity to ask questions and discuss the study?	**Yes**	**No**
Have you received satisfactory answers to all of your questions?	**Yes**	**No**
Have you received enough information about the study?	**Yes**	**No**
Do you agree that your participation in this study is voluntary and that you are free to withdraw at any stage?	**Yes**	**No**
Do you understand that the project has been reviewed by, and received ethics clearance through, the Research Ethics Approval Committee for Health of the University of Bath?	**Yes**	**No**
Do you understand who will have access to personal data provided, how the data will be stored, and what will happen to the data at the end of the project?	**Yes**	**No**
Do you agree to take part in the study?	**Yes**	**No**

Participant (Please print name)

Signature **Date**

Witnessed consent (please print name)

Signature **Date**

Researcher (please print name)

Signature **Date**

Appendix 17: Random allocation schedule

Neuropathic pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
MCR-NP1	Standard Physiotherapy + Walking			
MCR-NP2	Standard Physiotherapy ONLY			
MCR-NP3	Walking ONLY			
MCR-NP4	Standard Physiotherapy + Walking			
MCR-NP5	Standard Physiotherapy + Walking			
MCR-NP6	Standard Physiotherapy + Walking			
MCR-NP7	Standard Physiotherapy + Walking			
MCR-NP8	Walking ONLY			
MCR-NP9	Standard Physiotherapy ONLY			
MCR-NP10	Standard Physiotherapy + Walking			
MCR-NP11	Standard Physiotherapy ONLY			
MCR-NP12	Standard Physiotherapy + Walking			
MCR-NP13	Walking ONLY			
MCR-NP14	Walking ONLY			
MCR-NP15	Walking ONLY			
MCR-NP16	Standard Physiotherapy ONLY			
MCR-NP17	Standard Physiotherapy ONLY			
MCR-NP18	Standard Physiotherapy + Walking			
MCR-NP19	Standard Physiotherapy + Walking			
MCR-NP20	Standard Physiotherapy + Walking			
MCR-NP21	Standard Physiotherapy ONLY			
MCR-NP22	Walking ONLY			
MCR-NP23	Walking ONLY			
MCR-NP24	Standard Physiotherapy ONLY			
MCR-NP25	Walking ONLY			
MCR-NP26	Standard Physiotherapy ONLY			
MCR-NP27	Standard Physiotherapy + Walking			
MCR-NP28	Standard Physiotherapy ONLY			

Neuropathic pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
MCR-NP29	Standard Physiotherapy ONLY			
MCR-NP30	Walking ONLY			
MCR-NP31	Walking ONLY			
MCR-NP32	Standard Physiotherapy ONLY			
MCR-NP33	Standard Physiotherapy + Walking			
MCR-NP34	Standard Physiotherapy + Walking			
MCR-NP35	Standard Physiotherapy ONLY			
MCR-NP36	Walking ONLY			
MCR-NP37	Walking ONLY			
MCR-NP38	Walking ONLY			
MCR-NP39	Standard Physiotherapy + Walking			
MCR-NP40	Standard Physiotherapy ONLY			
MCR-NP41	Standard Physiotherapy ONLY			
MCR-NP42	Standard Physiotherapy + Walking			
MCR-NP43	Standard Physiotherapy + Walking			
MCR-NP44	Standard Physiotherapy + Walking			
MCR-NP45	Walking ONLY			
MCR-NP46	Standard Physiotherapy ONLY			
MCR-NP47	Standard Physiotherapy + Walking			
MCR-NP48	Walking ONLY			
MCR-NP49	Walking ONLY			
MCR-NP50	Walking ONLY			
MCR-NP51	Standard Physiotherapy ONLY			
MCR-NP52	Standard Physiotherapy ONLY			
MCR-NP53	Standard Physiotherapy ONLY			
MCR-NP54	Walking ONLY			
MCR-NP55	Walking ONLY			
MCR-NP56	Standard Physiotherapy + Walking			

Nociceptive pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
MCR-NC1	Walking ONLY			
MCR-NC2	Standard Physiotherapy ONLY			
MCR-NC3	Standard Physiotherapy + Walking			
MCR-NC4	Standard Physiotherapy + Walking			
MCR-NC5	Walking ONLY			
MCR-NC6	Standard Physiotherapy + Walking			
MCR-NC7	Standard Physiotherapy ONLY			
MCR-NC8	Standard Physiotherapy + Walking			
MCR-NC9	Walking ONLY			
MCR-NC10	Standard Physiotherapy ONLY			
MCR-NC11	Standard Physiotherapy ONLY			
MCR-NC12	Walking ONLY			
MCR-NC13	Walking ONLY			
MCR-NC14	Standard Physiotherapy + Walking			
MCR-NC15	Standard Physiotherapy + Walking			
MCR-NC16	Standard Physiotherapy + Walking			
MCR-NC17	Standard Physiotherapy ONLY			
MCR-NC18	Standard Physiotherapy + Walking			
MCR-NC19	Walking ONLY			
MCR-NC20	Standard Physiotherapy ONLY			
MCR-NC21	Standard Physiotherapy + Walking			
MCR-NC22	Standard Physiotherapy ONLY			
MCR-NC23	Standard Physiotherapy + Walking			
MCR-NC24	Standard Physiotherapy ONLY			
MCR-NC25	Walking ONLY			
MCR-NC26	Walking ONLY			
MCR-NC27	Standard Physiotherapy + Walking			
MCR-NC28	Standard Physiotherapy + Walking			
MCR-NC29	Standard Physiotherapy ONLY			
MCR-NC30	Walking ONLY			
MCR-NC31	Walking ONLY			
MCR-NC32	Walking ONLY			
MCR-NC33	Standard Physiotherapy ONLY			

Nociceptive pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
MCR-NC34	Standard Physiotherapy ONLY			
MCR-NC35	Standard Physiotherapy ONLY			
MCR-NC36	Standard Physiotherapy ONLY			
MCR-NC37	Standard Physiotherapy + Walking			
MCR-NC38	Standard Physiotherapy + Walking			
MCR-NC39	Standard Physiotherapy + Walking			
MCR-NC40	Standard Physiotherapy + Walking			
MCR-NC41	Standard Physiotherapy + Walking			
MCR-NC42	Standard Physiotherapy ONLY			
MCR-NC43	Standard Physiotherapy ONLY			
MCR-NC44	Walking ONLY			
MCR-NC45	Standard Physiotherapy ONLY			
MCR-NC46	Standard Physiotherapy ONLY			
MCR-NC47	Standard Physiotherapy + Walking			
MCR-NC48	Standard Physiotherapy ONLY			
MCR-NC49	Walking ONLY			
MCR-NC50	Walking ONLY			
MCR-NC51	Standard Physiotherapy ONLY			
MCR-NC52	Standard Physiotherapy + Walking			
MCR-NC53	Walking ONLY			
MCR-NC54	Walking ONLY			
MCR-NC55	Walking ONLY			
MCR-NC56	Walking ONLY			

Neuropathic pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
ABS-NP1	Standard Physiotherapy ONLY			
ABS-NP2	Standard Physiotherapy + Walking			
ABS-NP3	Standard Physiotherapy ONLY			
ABS-NP4	Walking ONLY			
ABS-NP5	Standard Physiotherapy + Walking			
ABS-NP6	Standard Physiotherapy + Walking			
ABS-NP7	Walking ONLY			
ABS-NP8	Standard Physiotherapy + Walking			
ABS-NP9	Walking ONLY			
ABS-NP10	Walking ONLY			
ABS-NP11	Standard Physiotherapy ONLY			
ABS-NP12	Standard Physiotherapy ONLY			
ABS-NP13	Standard Physiotherapy + Walking			
ABS-NP14	Standard Physiotherapy ONLY			

Nociceptive pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
ABS-NC1	Standard Physiotherapy ONLY			
ABS-NC2	Walking ONLY			
ABS-NC3	Standard Physiotherapy + Walking			
ABS-NC4	Standard Physiotherapy ONLY			
ABS-NC5	Walking ONLY			
ABS-NC6	Standard Physiotherapy ONLY			
ABS-NC7	Standard Physiotherapy + Walking			
ABS-NC8	Standard Physiotherapy + Walking			
ABS-NC9	Walking ONLY			
ABS-NC10	Standard Physiotherapy + Walking			
ABS-NC11	Walking ONLY			
ABS-NC12	Walking ONLY			
ABS-NC13	Standard Physiotherapy ONLY			
ABS-NC14	Standard Physiotherapy ONLY			

Nociceptive pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
DRT-NC1	Standard Physiotherapy + Walking			
DRT-NC2	Standard Physiotherapy + Walking			
DRT-NC3	Standard Physiotherapy + Walking			
DRT-NC4	Standard Physiotherapy ONLY			
DRT-NC5	Walking ONLY			
DRT-NC6	Walking ONLY			
DRT-NC7	Standard Physiotherapy ONLY			
DRT-NC8	Standard Physiotherapy ONLY			
DRT-NC9	Walking ONLY			
DRT-NC10	Walking ONLY			
DRT-NC11	Standard Physiotherapy ONLY			
DRT-NC12	Walking ONLY			
DRT-NC13	Standard Physiotherapy + Walking			
DRT-NC14	Standard Physiotherapy + Walking			

Neuropathic pain phenotype				
Study ID	**Treatment**	**Patient file no.**	**Physio**	**Date recruited**
DRT-NP1	Walking ONLY			
DRT-NP2	Standard Physiotherapy ONLY			
DRT-NP3	Walking ONLY			
DRT-NP4	Standard Physiotherapy + Walking			
DRT-NP5	Standard Physiotherapy + Walking			
DRT-NP6	Standard Physiotherapy ONLY			
DRT-NP7	Walking ONLY			
DRT-NP8	Walking ONLY			
DRT-NP9	Standard Physiotherapy ONLY			
DRT-NP10	Standard Physiotherapy + Walking			
DRT-NP11	Standard Physiotherapy ONLY			
DRT-NP12	Standard Physiotherapy ONLY			
DRT-NP13	Walking ONLY			
DRT-NP14	Standard Physiotherapy + Walking			

DRT - North-riding Private Practice Neurosurgical Rooms.

MRC - Randburg Medicross Clinic.

ABS - Doornfontein Towers West Medical and Dental centre.